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MINOR SURGICAL AFFECTIONS OF THE ANAL CANAL.

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IN 1888 the late Professor J. Symington renamed the old third part of the rectum, designating it a separate entity, the "anal canal"—a nomenclature which unfortunately has become firmly established throughout the various anatomical schools of the British Empire. Such an arbitrary subdivision is not only unscientific and unnatural, but from the surgical point of view it is definitely misleading, because it designates as one morphological entity two structures derived respectively from the post-allantoic gut and the proctodeal invagination, both of which have maintained their independent blood supplies, lymphatic drainage and innervation.

The importance of the anal canal may be judged from the observations of Rawson Pennington, who estimated that 85% of all ano-rectal diseases originated in this particular segment of the alimentary canal.

The purpose of this lecture is not only to discuss the anatomy of this particular area, but also to draw attention to the more important lesions with which it may become afflicted.

ANATOMY.

In the resting state the anal canal is a slit-like passage, about one and a quarter inches long, with its long axis directed downwards and backwards at about a right angle with that of the perineal portion of the rectum. During defæcation this segment becomes oval in shape and appreciably shorter.

When an examination is made of the interior of the canal, the lower fifth, except for the absence of hair, will be found to be indistinguishable from ordinary skin, whilst the upper three-fifths will be occupied by the columns of Morgagni or *columnæ rectales*, which are covered by columnar epithelium. Besides a terminal artery, each column of Morgagni contains a moniliform venous channel which on engorgement projects as a rudimentary pile. The intervening fifth or pecten, lined by smooth, glistening, transitional, stratified epithelium, lies between the wavy dentate or pectinate line and the white line (blue in the living subject) of Hilton, and corresponds to the interval between the subcutaneous external and internal sphincter muscles—that is, the anal intermuscular septum.

A series of irregular folds, the anal valves, connects the bases of adjacent columns so as to form small pockets known as the crypts or lacunæ of Morgagni, in which fæcal matter or small foreign bodies may lodge. These valves are the remnants of the broken-down anal or proctodeal plate, the so-called proctodeal hymen. Frequently small, white, shining nodules, from one-eighth to one-third of an inch long, are found on these valves or on the columns near the valves. Each one contains an artery and a small nerve trunk with a ganglionic enlargement at its base. These tags, known as the anal papillæ of Morgagni, are alleged to be important tactile organs through which a healthy person is warned of the presence of a fæcal mass, and the involuntary escape thereof is prevented. In addition, the lower two-fifths of the anal canal has an abundant nerve supply, which not only accounts for its extreme sensitiveness, but also explains

why operations involving this particular area are so exceedingly painful.

The pecten is a very important area marking the dividing line or physiological threshold separating the internal and external sphincter muscles, the somatic and visceral blood supply and lymphatic drainage, as well as the cerebro-spinal and autonomic innervation. Further, it is at this point that the main tracks of most fistulæ reach the lumen and either travel up the anal canal submucously, superficially to the muscles, or actually burst into the anal canal itself.

The pubo-rectalis, the best developed muscle of the pelvic diaphragm, forms a powerful sling supporting and anchoring the ano-rectal junction to the symphysis and adjacent pubes. This muscle, whose upper border may be identified on rectal examination, serves to mark the ano-rectal junction. The pubo-rectalis is innervated by branches of the pudendal nerve derived from the third and fourth sacral roots, whilst the remainder of the muscle receives its supply from the perineal branch of the fourth sacral nerve.

At the upper end of the anal canal the external muscular coat of the rectum becomes fibro-muscular and, after blending with the pubo-rectalis and deep part of the external sphincter, terminates by dividing into two or three fibro-muscular septa, which separate and ensheath the various segments of the external sphincter.

The outer lamella divides as it runs laterally to pass above and below the superficial external sphincter, whilst the inner division, passing downwards in the interval between the internal and subcutaneous sphincters, becomes the anal intermuscular septum and gains attachment to the skin of the anus just below the muco-cutaneous junction. Hence the origin of the sulcus so commonly seen in cases of intero-external prolapsed and thrombosed piles.

The internal sphincter is the direct continuation downwards of the circular muscle coat of the rectum and forms a tubular muscle encircling the upper three-fifths of the anal canal.

The external sphincter is a bilaminar or trilaminar muscle which, together with the pubo-rectalis portion of the *levator ani*, forms a thick ring of muscular fibres surrounding the anal canal.

The subcutaneous lamina, situated below but in the same plane as the internal sphincter, from which it is separated by the inner lamella of the tendinous expansion of the longitudinal muscular coat, is easily felt beneath the skin of the anal canal. These fibres, which encircle the lower fifth of the anal canal, decussate freely in front and behind the anus with their fellows of the opposite side. This lamina, which should not be confused with the non-striated *corrugator cutis ani*, is often erroneously referred to and diagrammatically depicted as constituting the whole of the external sphincter. The remainder of the muscle is placed on a supero-lateral plane so as to encircle the internal sphincter, and forms a strong supporting muscular cylinder.

The superficial or coccygeal lamina, forming the main part of the muscle, arises from the coccyx and ano-coccygeal raphe and is inserted into the central point of the perineum.

The deeper layer, continuous above with the pubo-rectal fibres of the *levator ani* and below with the superficial lamina, encircles and forms a complete sphincter for the anal canal. Upon this muscle the voluntary closure of the anal canal depends; hence its preservation is of the utmost importance to every patient. Normally it relaxes during defæcation and contracts as soon as the rectal contents are expelled. The muscle draws its nerve supply from two sources, namely, the perineal branch of the fourth sacral nerve and the inferior hæmorrhoidal branch of the pudendal nerve.

The pararectal space, containing perirectal fatty tissue and the lymphatic sinus of the rectum, lies between the longitudinal muscular coat and the *fascia propria*; the latter shuts it off from the superior pelvi-rectal space of Richet.

The superior hæmorrhoidal artery ends by dividing into a right postero-lateral and a left antero-lateral branch, both of which pierce the muscular wall of the rectum opposite the third sacral vertebra. The terminal branches of both divisions are important, not only because they occupy constant positions in relation to the anal canal, but also because of their relationship to internal hæmorrhoids. Further, as their pulsation may be felt by the examining finger, their exact location with reference to the circumference of the anal canal can be verified.

The right primary branch divides in the submucous coat into an anterior and a posterior branch, which course downwards respectively on the right anterior and right posterior quadrants of the anal canal.

The posterior branch gives off additional lateral and posterior twigs, which pass downwards on the right side of the anal canal and in the middle line posteriorly.

The left main branch runs down on the left wall of the rectum, but near its termination may give rise to anterior and posterior branches, which pass downwards in the left anterior and left posterior quadrants respectively.

Very rarely the anterior of these two branches gives off an accessory branch which descends in the anterior wall of the anal canal.

The main terminal branches of the superior hæmorrhoidal artery, together with their associated *venæ comites* and venous plexuses, take part in the formation of primary internal hæmorrhoids, which must consequently be situated in the right anterior, right posterior and left lateral aspects of the anal canal. Branches of the above terminal branches may, in a similar manner, give rise to secondary hæmorrhoids, which will be found on the left anterior or left posterior quadrants, on the right lateral wall or in the middle line posteriorly. Hence if all possible hæmorrhoids are developed, the pecten will mark the lower limit of three primary and five secondary hæmorrhoids.

The middle hæmorrhoidal vessels fail to furnish an adequate blood supply to the pecten, which therefore becomes dependent upon the anastomosis from above and below for its arterial supply and venous drainage. A minute capillary plexus lying in the submucous layer forms the connecting link between the superior hæmorrhoidal plexus above and the inferior hæmorrhoidal plexus below, and incidentally also between the portal and systemic circulations. Traced upwards, these small submucous veins join together and are joined by other veins from the surrounding parts to form larger and more tortuous veins, which ascend in the columns of Morgagni, where they usually present ampullary enlargements, varying in size up to that of a small pea; these are said to be the starting point of internal hæmorrhoids. Similarly the submucous veins of the pecten are apt to become dilated by constant straining, and when one is ruptured it forms the so-called thrombotic external pile, the more modern term for which is *hæmatoma ani*. The middle hæmorrhoidal plexus of veins lies on the outer side of the rectum immediately above the *levator ani* muscle.

PECTENOSIS.

Pectenosis, first described by Ernest Miles in 1918, is a local pathological condition in which a deposit of fibro-muscular tissue of varying breadth and thickness occurs in the submucous tissue of the pecten and leads to the development of a "pecten band", which encircles the anal canal like an india-rubber umbrella ring. This deposition of fibrous tissue is very similar to that occurring in cardiospasm and post-cricoid spasmodic stricture, and is alleged to be produced by hypertonus or other causes producing local passive congestion. For example, long-continued pressure upon the superior hæmorrhoidal veins by solid fæcal masses, as in dyschezia, will produce passive congestion of the submucous plexus of the pecten. With the passage of months or years the pecten band becomes more and more dense, and ultimately prevents proper relaxation of the external sphincter. Difficulty is then experienced in obtaining complete evacuation of the rectum, and the tighter the stricture becomes, the greater the straining at stool. When the condition is allowed to

progress, the anus may become almost completely closed and chronic intestinal obstruction may supervene. *Pruritus ani* is not an uncommon symptom in this condition.

It has been stated that the external sphincter is the only muscle in the body that does not relax under surgical anæsthesia; hence many surgeons, when operating in ano-rectal conditions, practise devulsion of the sphincter as a routine measure. The obstacle to relaxation is really the pecten band, and when this is divided the external sphincter will relax like all other voluntary muscles. However, pectenotomy is to be preferred to so-called "stretching of the sphincters", because no submucous areas of hæmorrhage will be left to fibrose and contract; the sphincter is at once restored to its full powers of relaxation.

The lower half of the anal canal is everted and a radial incision, usually at the four o'clock position, is made parallel to the long axis of the bowel and extending from the pectinate to the ano-cutaneous line.

Anal Fissure.

A fissure is primarily a linear tear in the epithelial lining of the anal canal. It overlies the subcutaneous external sphincter and the anal intermuscular septum, and rarely extends above this level. It is more common in men than in women, the percentage being about four to three. Owing to the relative lack of support of the posterior wall of the anal canal, the passage of a large hardened stool, superinduced by constipation, is prone not only to overdistend the canal, but also to cause a linear tear in the middle line posteriorly. The increased percentage of anterior fissures in women is attributed to relaxation of the perineum following parturition. With increasing chronicity the ulcer becomes deeper, the margins become indurated and the characteristic "sentinel tag" of Van Duren may form at its lower end.

In anal fissure the normal state of contracture of the external sphincter becomes exaggerated and the muscle itself soon becomes hypertrophied and indurated to such an extent that it is quite incapable of adequate relaxation. For this reason sphincterotomy may be frequently necessary in order that the tonic contraction may be temporarily suspended. When the lesion is of long standing the base consists of fibrous tissue which is the exposed "pecten band". The principal manifestation is pain just within the anus, of variable intensity and severity, which comes on after defæcation and may persist for several hours. In such circumstances defæcation is not infrequently unduly postponed, with the result that when the bowels do act the motion is hard and constipated, and the pain produced is much increased. Further, a localized abscess may form and discharge into the anal canal (for example, an internal subcutaneous sinus) or rupture externally, leaving a short dorsal subcutaneous fistula. The pain may also radiate to the back, perineum, testicles and bladder, or may simulate sciatica. Bleeding, except in the early stages, is not a usual feature, and a purulent discharge should always suggest a complicating submucous fistula.

The following is an illustrative case.

Mrs. J.B., aged thirty-five years, had suffered from frequent and painful micturition for five years; she had had three severe exacerbations in September, 1928, August, 1931, and March, 1932. She passed urine eight to twelve times during the day and three or four times at night, and suffered acute cutting pain at the termination of the act.

In 1928 examination at a metropolitan hospital disclosed a normal urinary system; cystoscopy revealed a normal bladder, and on ureteral catheterization the function of both kidneys was good. During August, 1931, micturition was occurring at fifteen-minute intervals (both by day and by night) and each act was attended by much pain. Cystoscopy was performed at a district hospital, a diagnosis of tuberculosis of the right kidney was made and nephrectomy was advised. A fortnight later cystoscopy was repeated at a metropolitan hospital, and except for the fact that a reddened area the size of a threepenny piece lay behind the left ureter, a normal bladder with normal ureteric orifices was disclosed. In a specimen of urine from the right ureter no pus and no acid-fast bacilli were found. No report of the ordinary urine examination was made available.

In March, 1932, a third exacerbation occurred, and it was decided to carry out a thorough examination under general anæsthesia. Vaginal examination revealed a normal uterus and adnexa, cystoscopy again revealed no abnormality, but rectal examination disclosed an indurated fissure with a pronounced "sentinel pile" in the middle line posteriorly. The sphincters were stretched and a linear incision was made through the base of the ulcer (Boyer's operation). On recovery from the anæsthetic the patient experienced almost instant relief of her symptoms, which until 1937 had not recurred.

Palpation of the anal margin will reveal spasmodic contraction of the subcutaneous external sphincter, and separation of the anal folds posteriorly will disclose the lower extremity of a linear ulcer.

A little cotton-wool twisted around the end of a probe and moistened with a 2% solution of "Percaine" or a 3% solution of benzocaine in oil is inserted into the anterior part of the anal canal and gently pressed backwards towards the fissure until such time as the lesion is rendered anæsthetic. A digital examination may bring to light certain other associated conditions, such as hæmorrhoids, a submucous fistula or a carcinoma, or may reveal certain signs characteristic of chronicity—induration of the lower border of the fissure or the presence of a pecten band.

Palliative treatment with sedative ointments, mild laxatives and bathing is likely to be successful only if the ulcer is superficial, impalpable and of short duration, and if the spasm of the sphincters is not pronounced. More recently the injection of quinine urea dihydrochloride (5%) or of "Proctocaine" [which is a 1·5% solution of procaine with butyl-p-amino-benzoate (6%) and benzyl alcohol (5%) in sterilized almond oil] posterior to, at the sides of and deep to the fissure, has been recommended, with the object of obtaining relaxation of the spasmodic contraction. The injection should give relief from pain for about a week, during which time the fissure may be expected to heal.

In the more chronic types of this malady, particularly if other associated lesions are present, recourse must be had to sphincterotomy (Boyer's operation) or stretching of the sphincters (Récamier's operation). The site chosen for sphincterotomy is at the four or eight o'clock position, because this site marks the junction of the anterior two-thirds of the muscle supplied by the inferior hæmorrhoidal nerve, and the posterior third, which is supplied by the perineal branch of the fourth sacral nerve. If a pecten band is present it should be divided through the same longitudinal incision. Boyer divided the external sphincter through the base of the fissure and then trimmed off the cutaneous edges with scissors so as to leave an open oval wound.

By either of the above operations the fissure obtains the physiological rest so necessary for repair. More recently excision of the fissure together with a triangular area (1·5 by 1·25 by 1·5 inches) of perianal skin, combined with posterior section of the subcutaneous external sphincter, has been advocated by W. B. Gabriel, of Saint Mark's Hospital, for the cure of fissure *in ano*.

Perianal Abscess.

All abscesses in connexion with the anal canal originate, with rare exceptions, from an infection through an abrasion in the pecten. The infection leads to a septic phlebitis or lymphangitis and a focus of localized suppuration develops in the perianal tissues. They differ in no important details from abscesses in other situations. There are five well-recognized varieties, namely: (i) anal (subcutaneous or marginal), (ii) submucous, (iii) ischio-rectal, (iv) pelvi-rectal, and (v) retrorectal (presacral).

An anal abscess lies between the subcutaneous external sphincter and the skin, whilst the submucous variety occurs between the mucous membrane and the internal sphincter.

An ischio-rectal abscess extends out between the laminæ of the external sphincter, fills the ischio-rectal fossa and becomes prominent beyond the lateral border of the external sphincter.

An anal abscess is obvious; but a submucous or ischio-rectal abscess, before attaining a large size, may easily be

overlooked, unless the index finger is introduced into the bowel and the surrounding tissues are palpated between it and the thumb.

Abscesses which form above the *levator ani* are fortunately rare. In this area there is a large potential space, the superior pelvi-rectal space of Richet, in which a considerable collection of pus may accumulate without giving rise to much discomfort to the patient and causing, except for signs of "septic intoxication", apparently insignificant symptoms.

The sequel to the majority of these perianal abscesses is a sinus or a fistula, and lack of appreciation of this fact has led to much discontent amongst patients. However, a considerable proportion of such fistulæ may be prevented, provided the abscesses which precede them are dealt with promptly and effectively.

It cannot be too strongly emphasized that a perianal abscess should be operated upon at once and not be subjected to expectant treatment by hot fomentations *et cetera* in the hope that it may "disperse" or burst. The patient must be prepared, when possible, in the same manner as for other rectal operations, and then placed in the lithotomy position. A low spinal anæsthetic has proved ideal in such cases.

An anal or an ischio-rectal abscess is customarily opened by a free incision radiating from the anus over the most prominent part of the swelling; but in the latter case an additional incision, at right angles to the first so as to form a crucial incision, will be required to open up the abscess cavity thoroughly. Four triangular pieces of skin will thus be formed, two-thirds of which should be cut away with scissors, the skin being held by the apices with toothed forceps. Fibrous septa running across the abscess cavity should be broken down with the finger and, as far as possible, the whole should be thrown into one large cavity. If the abscess contains gas or if surgical emphysema is present, gas-gangrene antitoxin should be injected.

The common cause of failure in the treatment of an ischio-rectal abscess is to make too small an incision to secure adequate drainage, whereupon the various pockets that are found in this condition extend and multiply.

A submucous abscess must be opened from within the bowel; the anus is dilated and a duck-bill speculum is inserted so as to expose the abscess freely.

As hæmorrhage may be troublesome after the customary surgical incision, the abscess is preferably opened by a diathermy knife. All overhanging edges of mucous membrane must be removed so as to lay the abscess cavity freely open, and then a moderate-sized drain tube is inserted into the anal canal and secured by stitches. An alternative procedure is to make a small nick into the upper part of the abscess, then pass a hooked Ryan's probe through the opening and pull until the end can be felt through the skin lining the pecten. Everything lying superficial to the probe is then incised and the abscess cavity will be freely opened up with very little damage to the mucous membrane.

In the treatment of pelvi-rectal and retrorectal abscesses an incision is made in the lateral part of the respective posterior quadrant of the perineum, and a director, guided by a finger in the rectum, is then passed upwards through the *levator ani* until pus is reached. A pair of sinus forceps is then passed along the director and the track through the muscle is stretched until it is large enough to admit a finger. It is advisable to use a large drainage tube in preference to packing the cavity with gauze.

In those cases in which the abscess cavity has extended below the pelvic diaphragm, treatment should be on the lines laid down for an internal pararectal sinus with an ischio-rectal extension.

ANO-RECTAL FISTULÆ AND SINUSES.

On November 18, 1687, Louis XIV of France was successfully operated upon by his *chirurgien en chef*, Monsieur Felix, for ano-rectal fistula, and having thus acquired royal patronage, this malady became a fashionable disease throughout the French Empire.

Although in a few cases a fistula forms without prior abscess formation—for example, as the direct result of a fissure—in the vast majority the fistula is the contracted cavity of a perianal abscess which has been inadequately treated and hence has failed to heal. The anatomical site of the original abscess, therefore, determines the type of fistula evolved.

These fistulæ are usually divided into three varieties: (i) complete fistulæ, (ii) blind internal fistulæ, and (iii) blind external fistulæ. The objection to this universally adopted classification is that the second and third groups are sinuses and not fistulæ.

The so-called complete fistula presents three characteristic features, namely: (i) the external opening, (ii) the main track, and (iii) the internal opening. The more deeply situated types of fistulæ, such as ischio-rectal and pararectal fistulæ, are prone to have secondary tracks and additional external openings. The external or perineal opening may be obvious, with pouting raised granulation tissue giving rise to a purulent discharge; but occasionally it is small, with no protruding granulations and admitting only a very fine probe. The main track is usually a firm, hard, fibrous tube, lined with soft granulations; the amount and density of the fibrous tissue depend upon the length of time during which the fistula has been in existence. The topography of the various tracks may be determined by the passage of a probe to determine the direction of the track or by palpation of the fibrous cord of indurations, as it runs through the pliable and elastic perineal tissues, by means of a finger in the anal canal and a thumb upon the skin surface of the perineum.

With the exception of the high-lying submucous or pararectal varieties, practically all fistulæ have their internal openings located in the region of the pecten. Hence the internal opening is best localized by light palpation of the pecten with a finger in the anal canal. It may be felt as an indurated irregular ulcer, as a hard fibrous dimple or as a rounded projection in the lining membrane. In point of fact, amongst American proctologists fistulæ are commonly classified into "anal" and "rectal" according to the relationship of the internal opening to the dentate or pectinate line. When the internal opening lies exactly on the pectinate line, the term "ano-rectal" is used.

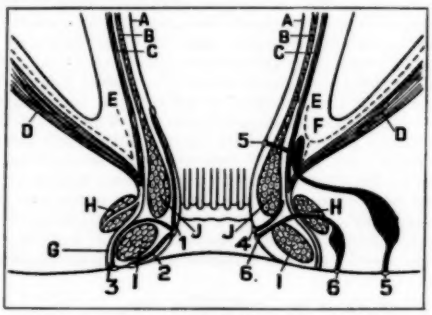

FIGURE I.

Diagrammatic representation of ano-rectal fistula. A: mucous membrane. B: circular muscular fibres. C: longitudinal muscular fibres. D: *levator ani*. E: pelvic fascia and *fascia propria*. F: pelvi-rectal space. G: tendinous insertion of longitudinal muscular fibres. H: deep portion of external sphincter. I: subcutaneous portion of external sphincter. J: internal sphincter. 1: submucous fistula. 2: subcutaneous fistula. 3: subsphincteric fistula. 4: intermuscular sinus. 5: pararectal fistula. 6: ischio-rectal fistula.

Owing to the inflammatory reaction which alters and obscures the anatomical outlines, it is not always easy to demonstrate the exact relationship of the fistulous track to the subcutaneous external sphincter; hence the muscle should be identified by palpation and followed in from an adjacent unaltered area.

The late Mr. David H. Goodsall, of Saint Mark's Hospital, London, was the first to point out that the internal and

external openings usually bore a definite relation to one another. Goodsall's rule stated that, if a line was drawn transversely across the centre of the anus, primarily external openings anterior to this plane usually had their internal openings immediately opposite to the external one—that is, in the same radial line—and such fistulæ were therefore straight and simple; but, on the other hand, when the primary external opening was behind the transverse anal line, the internal opening was usually found in the middle line posteriorly, and the main track was consequently more or less curved—in other words, a "semi-horseshoe". Although certain exceptions may be encountered, this rule has proved to be an accurate and valuable guide in operations for subsphincteric and ischio-rectal types of fistulæ.

The so-called incomplete fistula is really a sinus having a single orifice, which may open either onto a cutaneous surface or into the lumen of the bowel. The internal openings of anal and submucous sinuses, although often palpable, are sometimes best located by means of a proctoscope.

From an anatomical point of view the following classification of fistulæ may be adopted:

I. *Subcutaneous Type.*—The subcutaneous type is derived from an anal abscess and is located in the perianal subcutaneous tissue.
   (*a*) External subcutaneous sinus due to a suppurating *hæmatoma ani.*
   (*b*) Internal subcutaneous sinus, dependent upon and occurring in association with a fissure *in ano.*
   (*c*) Subcutaneous fistula, a further stage of (*b*) and able to be complicated by an internal submucous sinus.

II. *Submucous Type.*—The submucous type is derived from a submucous abscess and is confined to the submucous layer of the anal canal.
   (*a*) External submucous sinus, which generally originates from an infected submucous hæmatoma.
   (*b*) Internal submucous or rectal sinus, in which the internal opening may be situated anywhere along the main track, but is generally found in the upper part of the pecten.
   (*c*) Submucous fistula (uncommon).
   (*d*) Bilateral submucous fistula (internal horseshoe or circumferential), which has a T-shaped disposition, the horizontal parts of which usually lie deep to the hæmorrhoidal veins.

III. *Ischio-Rectal Type.*—The ischio-rectal type is derived from an ischio-rectal abscess.
   (*a*) External ischio-rectal sinus.
   (*b*) Internal ischio-rectal sinus.
   (*c*) Ischio-rectal (or semi-horseshoe) fistula.
   (*d*) Bilateral ischio-rectal (or posterior horseshoe) fistula.

IV. *Pelvi-rectal and Retro-rectal Sinus.*—No fistulæ are produced, as pus does not penetrate the *fascia propria.*
   (*a*) Pelvi-rectal sinus, a vertical track posterior to the transverse anal line and remote from the anus.
   (*b*) Retro-rectal (presacral) sinus, whose opening is located about one inch lateral to the tip of the coccyx.

V. *Pararectal Type.*—The pararectal type is derived from a pararectal abscess lying between the longitudinal muscular coat and the *fascia propria.* The internal opening is into the rectum instead of the anal canal.
   (*a*) Internal pararectal sinus with its internal opening above the internal sphincter.
   (*b*) Pararectal fistula, which ascends vertically to the opening in the bowel above the level of the *levator ani.*
   (*c*) Bilateral pararectal fistula, in which swellings in the ischio-rectal fossæ extend upwards beyond the level of the *levator ani* and encircle the rectum.

VI. *Subsphincteric Type.*—The subsphincteric type lies deep to the subcutaneous (external) sphincter muscle.
   (*a*) Internal subsphincteric sinus.
   (*b*) Subsphincteric fistula.
   (*c*) Bilateral subsphincteric fistula (anterior horseshoe fistula).

VII. *Intermuscular Sinus.*—The intermuscular sinus is very rare.
   (*a*) Internal intermuscular sinus, which lies between the circular and longitudinal muscular coats of the anal canal and rarely exceeds one inch in length.

Treatment.

Prior to any surgical intervention it is obligatory upon the surgeon to determine not only the topography of the main track, but also the exact position of the internal opening. It should be noted that spinal anæsthesia, by causing relaxation of the muscles of the anal canal, adds greatly to the difficulty of determining the relationship of the track to the musculature of the anal outlet.

From the operative point of view fistulæ may be conveniently divided into simple and complex varieties. A simple fistula will consist of an internal opening, an intermediate straight or curved track and an external opening. On the other hand, a complex fistula will have a tortuous track with possible multiple openings, and hence includes such varieties as horseshoe, circumferential *et cetera.* On rare occasions two or more simple fistulæ may exist in the same patient, each having a separate perineal opening.

The principle underlying the surgical treatment of a simple fistula is to lay open the main track from end to end, together with any secondary offshoots, so that an open wound is created which is then allowed to heal by granulation or secondary intention. Such measures, however, should not be applied to complex, bilateral ischio-rectal or pararectal fistulæ, otherwise serious impairment of the muscular control of the anal canal may result. With complex fistulæ operative treatment may advisably be carried out in two or more stages so as to avoid damage to the sphincter muscles or gross deformity of the anal outlet.

After the customary preparation the external sphincter is dilated and a blunt-pointed director is then introduced through the primary external opening and passed along the main track so as to emerge at the internal opening. The overlying tissues, including the subcutaneous external sphincter where the track passes deep to that lamina, are then completely divided so as to liberate the probe. The pyogenic membrane (granulation tissue) lining the track is scraped away, and all overhanging or undermined edges of skin are removed so as to leave a large, flat, open wound. A careful search must be made along the floor and sides of the exposed track for apertures leading to secondary tracks.

Before the wound is packed with ribbon gauze it is advisable to divide the floor with a linear or herring-bone (Salmon's back-cut) incision, with the object of allowing granulations to come through and hasten healing.

When the main track takes a curved course, it will in many cases be impossible for the probe to reach the internal opening. In such cases the track must be slit up in two or more distinct sections. The director is gently passed along the track as far as it will go, and when further progress is impeded the point is forced through the skin at this point. After the overlying tissues have been slit this manœuvre is repeated.

Care must be taken to make the final incision into the bowel radial to the anus, otherwise the external sphincter may be divided obliquely and serious incontinence of fæces may result.

After fistulotomy an interval of five or six weeks—often considerably longer—must elapse before the wound becomes soundly healed, and during the greater part of this period the patient should be kept in a recumbent position.

From time to time surgeons have recommended excision of the fistula and primary suture of the resulting wound; but this method, whilst ideal on paper, is seldom satisfactory owing to the great difficulty in securing primary union of a wound in this situation.

Special Fistulæ.

Submucous Fistula.

The track of the submucous fistula not uncommonly takes a circular or curved course under the mucosa, hence comes into close relation to the hæmorrhoidal vessels. The treatment of a short fistula does not differ from that already described for a submucous abscess. However, in the case of a high-lying fistula it is frequently difficult to control the hæmorrhage, especially if the track pursues an oblique course; hence it may be more prudent to adopt

the old-fashioned method of destroying the mucosal roof with strangulating silk ligatures.

A probe, which is inserted at the lower opening, is made to traverse the main track and is then pushed through the mucous membrane covering the apex of the cavity. Two stout silk ligatures are threaded through the eye and the probe is withdrawn. The ligatures are then firmly tied without interlocking, so that the intervening strip of mucosa will necrose and freely open up the track from end to end.

Bilateral Ischio-Rectal or Posterior Horseshoe Fistula.

Although ischio-rectal fistulæ are prone to have multiple external openings, there is as a rule only one internal opening situated in the middle line posteriorly and lying between the external and internal sphincters. The barrier interposed by the origin of the superficial lamina of the external sphincter from the ano-coccygeal raphe prevents the initial abscess from pointing posteriorly; hence the pus tracks forwards around the anus to form lateral perineal openings.

All existing tracks on both sides, as well as the track passing beneath the ano-coccygeal raphe, should be laid completely open; but that part of the main track which passes through the external sphincter to the internal opening should not be incised until such time as the greater part of the wound has become filled in—that is, about three weeks later. By such a two-stage procedure there will be far less retraction of the divided ends, owing to the muscle fibres' becoming bound down by the new fibrous tissue.

To mark the track for the second stage of the operation a stout silk ligature may be knotted loosely around the external sphincter—a procedure which further aids in preventing the retraction of the divided muscle fibres.

When a deep secondary track, extendng forwards beneath the inferior hæmorrhoidal vessels, is discovered, the parts superficial to the vessels should be kept intact. If this track is laid open in the customary manner, the nerve supply to two-thirds of the external sphincter (on one side) will be divided and partial incontinence of fæces may result.

J.W., a male patient, aged thirty-eight years, who presented pronounced signs of septic intoxication, was seen in consultation on July 14, 1937, on account of severe perineal pain associated with a purulent rectal discharge of thirteen days' duration.

On examination a large, brawny, inflammatory swelling was found to occupy the left ischio-rectal fossa and to extend to the posterior part of the anal verge. On rectal examination an abscess could be felt lying behind the anal canal and communicating with its lumen in the middle line posteriorly through an opening in the pecten.

Under ethyl chloride anæsthesia a free crucial incision was made over the most prominent part of the swelling, about one and a half ounces of thin stinking pus were evacuated and the wound was packed with gauze. Fourteen days later, when the acute inflammation had subsided, a general anæsthetic was administered and a thorough examination of the rectum was carried out with the following result.

On the left side was a semi-horseshoe fistula running back to the dorsal abscess, which communicated anteriorly with the bowel through two openings. From the upper of these two openings a sinus ran round each side of the rectum parallel to and just above the level of the *levator ani* muscle, whilst below the track terminated in the semi-horseshoe fistula first mentioned.

This, then, was a case of bilateral pararectal fistula having two internal openings and one external opening. A two-stage operation was therefore planned. At the first stage the semi-horseshoe fistula was laid freely open to the point where it joined the vertical limb of the pararectal fistula beneath the ano-coccygeal raphe, which structure was itself divided. The track running through the pelvic diaphragm was dilated by Hegar's dilators and the index finger, aided by short incisions both dorsally and laterally (coccygeus muscles), and a moderate-sized rubber tube was inserted in order to secure adequate drainage. A stout silk ligature was passed along that part of the track traversing the external sphincter muscle and the two ends were securely tied.

At the second operation, six weeks later, the subcutaneous lamina of the external sphincter was divided radially by the laying open of that part of the track which had been marked by the silk ligature.

Pararectal Fistula.

Although an internal pararectal sinus may resemble a high-lying submucous track in producing a globular swelling, encroaching upon the lumen of the bowel, the mucous membrane in the former instance will be freely movable over the swelling. The surgical treatment of this type of sinus differs but little from that described for the drainage of a pelvi-rectal abscess.

On the other hand, with a pararectal fistula the external or perineal opening will be found to be about one and a half to two inches lateral to the anus, and if a probe is inserted it will pass upwards and inwards through the *levator ani* to enter the lateral wall of the rectum about one inch or more above the level of this muscle. The ischio-rectal fossa should be freely opened by a crucial or T-shaped incision. With a finger in the rectum to act as a guide, the aperture in the *levator ani* is located and enlarged in the direction of the muscle fibres. The extent of the cavity in the pararectal space is then determined and free drainage is established.

The floor of the ischio-rectal fossa is liberally removed, a wide, funnel-shaped wound being left from the floor to the roof of the fossa. When a pararectal fistula is bilateral, the track running to the supradiaphragmatic portion passes between the coccygeus muscles in the middle line posteriorly; hence these muscles must be divided for a short distance on either side in order to secure adequate drainage. If a second internal opening is found between the sphincters in the middle line posteriorly, the track lying beneath the external sphincter should be marked by a silk ligature and its opening up deferred to a subsequent operation.

Subsphincteric Fistula.

The subsphincteric fistula lies deep to the subcutaneous sphincter muscle, and its extension outwards into the ischio-rectal fossa is prevented by the lateral fibrous expansion of the longitudinal muscle coat as it passes beside the subcutaneous lamina of the external sphincter to gain insertion into the perianal skin. In other words, the primary external opening lies about one and a quarter inches from the anal verge, and in this the subsphincteric fistula differs from both the pararectal and the ischio-rectal types of fistula.

When the primary external opening lies anterior to the transverse anal line, the treatment is on the lines laid down for a simple fistula. On the other hand, when the opening lies posterior to this plane, those methods advocated for a semi-horseshoe fistula should be adopted. In either case a careful search for an accessory submucous track or an internal intermuscular sinus should be made. Any submucous extension must be treated on similar lines to those laid down for submucous sinuses.

Intermuscular Sinus.

Intermuscular sinuses resemble submucous tracks, from which they are difficult to distinguish because of the surrounding inflammatory thickening. The orthodox treatment for such sinuses is very similar to that adopted for a submucous sinus.

A director is inserted through the internal opening and after traversing the whole length of the sinus is forced through the wall of the bowel into its lumen. The tissues superficial to the director, which include the internal sphincter, are then severed, and thus the sinus track is freely laid open into the lumen of the bowel. Severance of the internal sphincter in the above manner has no effect on anal function, as the ano-rectal ring remains intact.

Tuberculous Fistula.

Tuberculosis is rarely primary in ano-rectal fistulæ, being secondary to tuberculosis elsewhere; hence tuberculous fistula is most commonly found in patients with

evidence of acute or arrested tuberculosis. The incidence varies from 3% to 20% in accordance with the incidence of tuberculosis amongst the population.

The perineal opening is usually large and irregular in shape, with undermined edges, and there is a thin watery discharge. The surrounding skin is of a reddish-purple tint, and induration of the main track is noticeably absent.

Operative measures should be confined to simple drainage, because healing will be unable to occur unless the patient's general resistance to tuberculosis is raised to a high level.

Ten Commandments for Fistulotomy.

1. Never lay open a fistula when an acute abscess is present. Be content with draining the abscess.
2. Provide good, free and thorough drainage to all parts of the fistulous track, the wound being so planned that the drainage will remain efficient until the parts have healed. If this is not possible owing to the size or depth of the tracks, the operation should be performed in two stages.
3. Never fail to inspect carefully the floor and sides of the main track for apertures leading to secondary tracks. The overlooking of a secondary track will lead to a recurrence.
4. Do not divide both external and internal sphincters at the same operation; incontinence of fæces is almost certain to result.
5. Never divide the external sphincter at more than one site in any particular operation, otherwise serious incontince of fæces may result. With very rare exceptions, there is only one internal opening to each fistula.
6. Never divide the external sphincter obliquely to its fibres, but only at right angles—that is, radial to the anus. An oblique division may result in an incontinent anus.
7. Perform in two stages the operation for a complex fistula (for example, posterior horseshoe or bilateral ischio-rectal) if it involves division of the external sphincter. At the first operation all tracks should be incised and freely laid open, except that which passes into the rectum through the external sphincter. At the second operation, which is generally performed three weeks later, this track is opened up.
8. Never operate upon a patient with a fistula unless you are prepared to watch and supervise the after-treatment. The after-treatment is nearly as important as the operation.
9. Never pack or plug a fistula wound. Packing or plugging with gauze prevents the growth of granulation tissue and delays the process of repair. Dressings must always be changed at least twice daily.
10. Keep the patient in bed until the wound is quite healed.

HÆMORRHOIDS.

Hæmorrhoids, or piles, are swellings produced by pathological changes in the veins supplied to the anal canal. According to their original location they are classified as external if situated at the anal margin and covered by skin, and internal when entirely within the anal canal and clothed by mucous membrane. There is a third variety, in which both an external and an internal pile coexist and in which the intervening lining membrane is prolapsed and made prominent by infiltration of the underlying connective tissue.

External Hæmorrhoids.

In the current text-books of surgery three separate and distinct conditions are described as external hæmorrhoids: (*a*) cutaneous piles, redundant folds of perianal skin, (*b*) *hæmatoma ani* or thrombotic external piles, and (*c*) dilated perianal veins.

Cutaneous or "fleshy" piles are an exaggeration of the normal perianal rugæ, and if not inflamed cause little inconvenience. Retained secretion may, however, collect between adjacent folds, causing irritation and inflammation, and leading to gradual increase in size and loss of elasticity of the rugæ—an appearance which from its resemblance to a dried fig is termed "*marisque*" by French authors. Further, after defæcation, difficulty may be experienced in cleansing the perianal skin, and fæcal matter tends to lodge between the folds and cause additional irritation and discomfort. When piles of this type are causing itching and discomfort they should be snipped off with scissors under local anæsthesia.

There is a difference of opinion as to the exact pathology of the so-called thrombotic external pile. These are acutely painful, rounded or oval swellings, varying in size from a pea to a marble, dark blue in colour and situated at the anal margin. The lump is caused either by a thrombosis in a dilated vein or, most commonly, by rupture of a small vein into the surrounding connective tissue. After the extravasated blood coagulates, the surrounding connective tissue becomes condensed by round-celled infiltration into a capsule. In the past this capsule has been mistaken for the wall of a dilated vein; hence the name "thrombotic pile". In treating this condition, if there is but a small tumour, it is necessary only to snip off the summit with a pair of scissors and express the clot. The skin edges fall together, and any bleeding is usually readily controlled by pressure. If the patient will not submit to this little operation, suppuration may necessitate an incision to prevent the formation of a sinus or fistula; but more usually the lump slowly subsides after treatment with anodynes, leaving a redundant perianal tag.

In the third variety the superficial veins surrounding the anal margin become dilated and during straining form a distinct swelling; when straining ceases the veins almost completely subside, leaving the skin loose and redundant. Dilated perianal veins or varicose external piles give rise to little inconvenience, beyond a feeling of fullness and uneasiness after defæcation. The condition is always associated with constipation and is frequently accompanied by internal hæmorrhoids. When a finger is introduced into the anal canal the extent of any hypertrophy or spasm of the sphincter as well as the presence of any complicating pecten band may be readily detected. If the administration of mild laxatives does not give relief, recourse should be made to sphincterotomy or pectenotomy, or both.

Internal Hæmorrhoids.

Owing to the fact that the veins of the portal system are not supplied with valves, internal hæmorrhoids are one of the penalties to be paid for the assumption of the upright position. This malady is not only the most common disease of the anal canal, afflicting about 80% of all people who have reached the fourth decade of life, but is often the primary cause of other disturbances.

An internal hæmorrhoid is a vascular tumour consisting chiefly of varicose veins accompanying a tortuous and dilated terminal branch of the superior hæmorrhoidal artery as it lies in the submucosa in the base of a column of Morgagni. In other words, an internal pile consists of a conglomeration of blood vessels—arteries, capillaries and veins—lying in the submucous tissue of the anal canal, which, owing to degeneration of their coats, have become enlarged and tortuous. It therefore follows that the classification of internal hæmorrhoids into arterial and venous has no underlying anatomical or pathological basis. There is, however, an uncommon variety, called a nævoid or capillary pile, in which the swelling consists of thinned mucous membrane covering a mass of capillaries.

When pathological changes take place in the arteries and veins, rendering them tortuous, dilated and thickened, the main terminal branches of the superior hæmorrhoidal vessels become affected first; hence the three primary hæmorrhoids, as already pointed out, must be situated on the right anterior (eleven o'clock), the right posterior (seven o'clock) and the left lateral (three o'clock) aspects of the anal canal. The four possible secondary hæmorrhoids, although closely connected above with the primary piles from which they originate, occupy intermediate positions between the primary hæmorrhoids.

During defæcation the anal mucosa is everted and the developing pile becomes engaged in the grip of the external sphincter muscle, receding after the act. This

constantly recurring drag leads to enlargement and progressive elongation of the pile, partly owing to increasing dilatation of the component vessels and partly to inflammatory exudation into the areolar tissue lying between the vessels. Hence internal hæmorrhoids are progressive in their development, and it is important to note that there are three distinct stages in the development of an internal pile. This does not mean, however, that in any given case all the piles will be in the same stage of development, because such an occurrence would be exceptional.

Stages in Development.

First Degree.—The pile is small in size and covered by healthy and unaltered mucosa, and its presence is characterized by recurrent hæmorrhage, often profuse, of bright red blood, which accompanies each act of defæcation. At this stage (so-called arterial pile) the pile does not protrude through the anal orifice, nor is it palpable on digital examination.

Second Degree.—The pile increases in bulk and becomes sufficiently elongated to protrude through the anal orifice during defæcation; but as soon as the effort is over it becomes spontaneously reduced. Owing to the thickening of the mucosal covering the pile loses its bright red colour; hence bleeding becomes less frequent and less profuse than in the preceding instance. At this stage the pile can be detected, on rectal examination, as a thickened longitudinal fold and may be made to protrude at the anus on the patient forcibly straining down.

Third Degree.—The piles protrude persistently through the anal orifice. They have to be replaced manually after each act of defæcation, and prolapse tends to recur on the slightest exertion—for example, coughing, sneezing *et cetera*. These piles (so-called venous piles) are tough and fibrous, considerably increased in size, and are covered by a thickened bluish-purple mucosa; hence bleeding becomes a rarity.

Symptoms.

Hæmorrhage occurs in over three-quarters of all cases of internal hæmorrhoids. It generally takes place during the act of defæcation, when the pile becomes gripped by the external sphincter. The hæmorrhage is rarely profuse, but the daily loss of blood is so constant that in time it may produce a state of profound anæmia. D. H. Murray reported a fatal case in a man, aged thirty-one years, in which the hæmoglobin value was only 10% and the red blood cells numbered 1,000,000 per cubic millimetre. I have seen the hæmoglobin value as low as 40% in this malady, and within eight weeks of operation the reading was restored to 84%. Prolapse is a late symptom of hæmorrhoids. As soon as the pile commences to protrude through the sphincter it is liable to suffer injury from friction or to become strangulated by the sphincter muscle. A mucoid discharge from the bowel is a common accompaniment of third degree hæmorrhoids.

Complications.

Strangulation.—If hæmorrhoids are forcibly protruded through an anal canal whose expansibility is limited owing to the presence of a pecten band strangulation, ulceration or even sloughing may supervene.

Fibrosis.—If an internal hæmorrhoid becomes thrombosed it is gradually converted into a fibrous pile, which may be sessile, or through continued traction may acquire a pedicle (fibrous polypus).

Secondary Anæmia.—Anæmia is a most common complication of internal hæmorrhoids and a slight degree of anæmia is a customary accompaniment.

Treatment.

Treatment on palliative lines by astringent medicaments, suitable laxatives and an appropriate dietetic régime *et cetera* may be recommended for certain first degree internal hæmorrhoids, especially those cases associated with portal or rectal congestion, as well as for piles complicated by inflammation and sepsis. Apart from these indications, since a cure cannot be effected by palliative measures, there is but little advantage in pursuing such a line of treatment. Prolapsed thrombosed piles should be reduced and kept in position by a firm perineal pad, and the patient should be confined to bed for a few days. The principal means of radical relief are five in number, namely: injection, ligature with incision, clamp and cautery, excision and suture, and excision of the so-called "pile-bearing" area.

Injection.—Injection, first introduced by Blackwood in 1866, is especially suitable for small uncomplicated first-degree piles. The objective is to cause a thrombosis of the vessels of the pile, and for this purpose a large number of solutions have been used. Most proctologists prefer a solution of quinine and urea hydrochloride or of phenol. The former is usually of 5% strength, whereas phenol is used in strengths ranging from 5% to 20%.

Bleeding from a hæmorrhoid is usually stopped as if by magic after an injection of a 5% phenol solution in almond oil. The injection in this case must be made below the mucosa (submucous injection), but above the hæmorrhoid. If one desires to inject the solution directly into the centre of a large vascular hæmorrhoid it is necessary to use a solution of at least 10% strength. It is generally advised not to inject more than one or two piles at each sitting, hence treatment must be protracted; furthermore, relapse is not very uncommon. The larger and more easily prolapsed the hæmorrhoid, the poorer the response to injection therapy. It is dangerous to inject hæmorrhoids which are thrombosed, infected or ulcerated, or when inflammatory conditions, such as abscess, fistula *et cetera*, complicate the piles.

Urgent indications for operation are two in number: copious and recurring hæmorrhage and uncontrollable protrusion. It does not appear to matter greatly which particular method is adopted, provided that the operation is performed efficiently. Whatever operation is performed, the sphincters should first be gently dilated.

As regards anæsthesia, an intravenous injection of soluble "Hexobarbitone" or an intrathecal injection of "Percaine" has proved to be both popular and efficient.

Ligature with Incision.—Ligature is the safest and best operation in the great majority of cases of internal hæmorrhoids. The technique was devised by the late Mr. Frederick Salmon, the founder of Saint Mark's Hospital, and has been regularly practised at that institution for over a century. Each hæmorrhoid is seized with pile forceps and a V-shaped incision is made with scissors in the muco-cutaneous junction at its base so as to open up the submucous space, care being taken not to include any muscular tissue. The pile is then stripped up by blunt dissection until it remains attached only by its upper pole, the source of its blood supply. An oblique cut is made on either side of the neck of the pile to narrow the pedicle, which is then crushed by a powerful pair of forceps. A stout strand of plaited silk (number 10) is placed in the groove formed by the separation of the pile, and the ligature is thereupon tightly tied as high up the pile as possible, with the knot on the bowel aspect—that is, "on the lumen". If the distal two-thirds of the pile are to be removed, great care must be taken to ensure that sufficient pedicle is left to prevent the ligature from slipping. When all the hæmorrhoids are thus ligated, they should be returned within the sphincter; after this is done, any redundant perianal skin that remains may be excised. The ligatures usually come away at about the eighth day, after which a granulating surface is left which gradually heals and contracts.

A modern innovation is to transfix the pedicle of the pile with number 2 chromicized catgut on a round-bodied needle and then to ligate the pile with a Staffordshire knot. To reduce the size of the bare area and hasten healing, the ligated stump may be brought down to the anal margin by a catgut suture (Sir C. Ball's modification). The development of inflamed and œdematous perianal tags may be largely prevented by adequate preparation and by the application of firm dressings subsequent to operation.

The Clamp and Cautery Operation.—The clamp and cautery method, originated by J. H. Cusack, of Dublin, in 1843, is suitable only for larger and more obvious hæmorrhoids; hence recurrence is not unusual, as the smaller secondary piles are frequently left untouched. Its chief value is in cases of inflamed, gangrenous or sloughing piles. Each pile is drawn down by pile forceps and its base is then enclosed within the blades of a special hæmorrhoidal clamp, which is screwed up tightly in the long axis of the bowel. The orthodox clamp has the blades covered with ivory to protect the surrounding tissues, but wet gauze can be used for the same purpose with any clamp. The pile is then cut off about one-eighth of an inch distal to the clamp, and the stump is thoroughly charred with a cautery heated to a dull-red heat. It is important to make sure that skin is not included within the grasp of the clamp, otherwise its cauterization will give rise to considerable post-operative pain.

After three minutes the clamp may be very gently relaxed, and if no bleeding occurs it is gently slid off so as not to open up the wound. Should bleeding occur, however, the clamp must at once be retightened and the cautery reapplied.

Excision and Suture.—Of the various modifications of the operation of excision and suture, the most popular is that described in 1903 by Mr. A. B. Mitchell. The base of each pile is clamped in the long axis of the bowel with a narrow-bladed clamp, and its upper pole, including the main vessels, is transfixed and ligated off just above the end of the clamp. The projecting hæmorrhoid is cut off and the edges of the mucous membrane are sutured; the clamp is thus sewn up with the included stump by means of a continuous suture. The clamp is now removed and the suture is pulled tight and tied towards the anal margin.

Whitehead's Operation (1887).—Whitehead's operation is the operation of election for internal hæmorrhoids complicated by considerable prolapse of the anal mucous membrane. An incision is made circumferentially through the lining of the anal canal, at or just above the mucocutaneous junction. The incision is deepened until the submucous space is opened up and the subcutaneous external sphincter muscle is defined. The whole of the mucous lining of the anal canal together with the hæmorrhoidal mass is gently separated by blunt dissection from the underlying structures until the healthy mucosa above is reached. The cuff of diseased mucosa is now removed and the healthy cut edge is stitched to the skin of the anal margin by catgut sutures.

If healing by primary union does not occur the mucosa is prone to recede and stenosis of the anal canal is apt to develop. Further, some temporary loss of anal sensation and a certain amount of rectal insecurity not uncommonly follow this operation.

In the past there has been much discussion on the relative merits and demerits of this operation; hence this procedure should be the prerogative of those experienced in rectal surgery.

CHANGES IN MUSCLE PRODUCED BY NERVE DEGENERATION.[1]

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IT is impossible in the course of one lecture to cover the whole field of investigations on the reaction of muscle to denervation (see the recent review by Tower[15]). Attention will largely be restricted to those aspects which relate to recent work at the Kanematsu Institute. This work is as yet unpublished, and I wish to thank my collaborators, Dr. Katz and Dr. Kuffler, for generously allowing me to refer to it.

[1] Read at a meeting of the Section of Neurology and Psychiatry of the British Medical Association on October 30, 1940.

In considering the problem of muscle degeneration one is first drawn to the well-known degeneration which occurs in any part of a cell separated from the nucleus. There are the standard illustrations showing this in microdissection studies on the protozoa-paramœcium or vorticella. It is also well shown in the degeneration of peripheral nerves, both when severed from their cell bodies in the central nervous system and ganglia, and when the cell body is killed by such diseases as progressive muscular atrophy and poliomyelitis. For example, in mammals peripheral nerve ceases to conduct impulses at about three to four days after severance, and at about the same time histological changes appear—disintegration of the axon and destruction of its membrane. The cause of this striking degenerative change is not known, but three negative observations have been made. (i) The degeneration is not due to disuse, for it is, if anything, slightly quickened if the nerve is artificially stimulated,[3] and it does not occur when there is prolonged and total disuse of nerve fibres connected with their nerve cells.[32] (ii) The degeneration does not appear to spread peripherally from the cut end; all parts of the nerve are affected simultaneously.[12] (iii) The distances along the length of the fibre are so great that the quick course of degeneration cannot be due to the cutting off of a supply of nutritive materials diffusing along the nerve fibres.

Some more subtle influence (called the trophic influence) must be at work within the confines of the living cell. It is dependent presumably on patterns of molecular orientation, and is responsible for integrating the diverse parts of the cell into a living unity. Degeneration occurs when this trophic influence is removed by severing a part of a cell from its nucleus.

When nerve degenerates, the changes which are produced in muscle have also been regarded as due to removal of a trophic control; but it is important to distinguish between this trophic control and the above trophic influences exerted within the confines of a cell. Furthermore, it will be seen that many of the changes observed in degenerating muscle occur also in muscle which is simply suffering from disuse, all its nervous connections being intact. This condition of "disuse atrophy" will be considered first before I go on to the more complex lesion shown by muscle when its motor nerve degenerates.

Disuse Atrophy.

Disuse atrophy is best demonstrated when a region of spinal cord has its dorsal roots cut, and is severed from the rest of the central nervous system. Provided that the circulation has not been badly damaged, the anterior horn cells and their motor nerve fibres live for months, and show no obvious change from normal.[33] However, with this cutting off of all incoming impulses, there is no discharge of impulses from these anterior horn cells and the muscles atrophy rapidly under this experimental condition of total disuse,[34] their weight and contraction strength being halved in as little as three weeks.

A similar rapid atrophy occurs after tenotomy,[9][18] though it is not known whether tenotomy is accompanied by complete disuse of the muscle. It has even been suggested that the degeneration of tenotomized muscle is due to over-activity.[18] It is hoped to settle this question by making electrical recordings from tenotomized muscle, and by stimulating it artificially in the manner described below for disuse atrophy.

In disuse atrophy the muscle fibres show a general atrophic change.[14] Both the muscle fibres and their subsarcolemmal and sole-plate nuclei diminish in diameter and stain more feebly; at the same time the interstitial fibrous tissue proliferates, and eventually largely replaces the muscle tissue. The physiological and pharmacological properties of this atrophic muscle show no gross changes, but have not yet been studied carefully.

As disuse atrophy results from the absence of muscle contractions normally induced by nerve impulses fired down the motor nerve fibres, it should be prevented by a certain duration of nerve tetanization. We are at present investigating the effects of different durations of this tetanization. Disuse atrophy is produced in both hind

limbs of the cat by physiological isolation of the lumbar and sacral regions of the spinal cord, as described above, and then different daily doses of maximal tetanic stimulation are applied to one sciatic nerve. Circulation to the contracting muscles is ensured and fatigue is minimized by employing intermittent stimulation—alternate stimulation and rest periods each of about one-half second. Our preliminary experiments have indicated that disuse atrophy may be prevented by a daily intermittent tetanization of only thirty seconds' duration. Further work is in progress in order to determine the minimum dosage of effective stimulation, the effects of much more prolonged stimulation, and any variations in dosage for muscles of different type—flexors or extensors; the slow postural or the fast "phasic" muscles. The effect of passive alternating movements and of massage on disuse atrophy will also be investigated.

The very short effective dosage of artificial stimulation suggests therapeutic possibilities. Clinically disuse atrophy is very common, occurring after a few days' rest in bed, and particularly if a limb is immobilized, for example, in plaster. If a few seconds' nerve stimulation is also adequate to prevent this atrophy in man, it should be possible to keep limbs in good condition even during prolonged immobilization.

Effects of Nerve Degeneration on Muscle.

In addition to the above effects observed in disuse atrophy, the following changes are also observed when nerve degenerates. In mammals they first appear at about the fourth day—that is, soon after the nerve becomes inexcitable—and gradually increase for some two weeks.

Hypersensitivity to Drugs such as Acetylcholine and Nicotine.

The muscles eventually become enormously more sensitive to such drugs as acetylcholine and nicotine, contractions being produced by doses 1,000 to 10,000 times smaller than normal. In addition mammalian muscles come to resemble the normal frog's muscle, in that with the acetylcholine response the initial contraction set up by muscle impulses is followed by a prolonged contracture during which no muscle impulses can be set up.[1] We have investigated this hypersensitivity by direct applications of acetylcholine to localized regions of the frog's sartorius. Normally the region of the motor nerve endings is at least ten times more sensitive than the rest of the muscle fibre. After denervation the sensitivity of all parts of the muscle is greatly increased, and the differential sensitivity is still observed.

Spontaneous Fibrillation.

The fine fibrillating movements of denervated muscle are due to contractions set up by impulses spontaneously arising in single muscle fibres.[1,7,9,10] At the Kanematsu Institute we have investigated this fibrillation in cat's muscles, using fine electrodes for leading from small bundles of muscle fibres. It has thus been shown that in a single fibre the rhythm of discharge is about ten per second. Further, electrical recording at different points along the length of the fibres shows that this rhythmic discharge is set up at a focus often widely separated from the region of the motor nerve ending on the muscle fibre, and from this focus the impulses are propagated normally along the whole length of the fibre. It was surprising to find that the fibrillation was not affected by curare and possibly only a little increased by eserine.

Contracture following Direct Stimulation.

Direct electrical stimulation of denervated muscle has long been known to be followed by a contracture. Rosenblueth and Luco[10] observed an increase in muscle impulses during this contracture, and by recording from narrow strips of denervated cat's muscle we have been able to show that this contracture is entirely due to the repetitive discharge of impulses resembling in every way the fibrillation; for example, their frequency is about ten per second and they begin anywhere along the muscle fibres. The direct stimulation of the muscles has induced a temporary fibrillation (from a fraction of a second up to several seconds in duration) in many muscle fibres not normally fibrillating.

Other Effects of Nerve Degeneration.

There is possibly an initial phase of activity and even proliferation of the nuclei, both those at the end-plate region and the subsarcolemmal nuclei along the whole length of the muscle (Tower[12]). However, degenerative changes resembling those observed with disuse atrophy soon preponderate.

Discussion.

The increased drug sensitivity and the fibrillation with its related contracture response both indicate an increased sensitivity of the surface membrane of the muscle fibres, and it has recently been suggested[1] that the fibrillation is due to the stimulating action of small amounts of acetylcholine. However, this seems to be excluded by our observation that curare greatly depresses the sensitivity of the denervated muscle to acetylcholine while not affecting the fibrillation. Also we have seen that fibrillation is due to impulses arising anywhere along the muscle fibre, and not only at the region (the end-plate zone) specially hypersensitive to acetylcholine. Thus there is no simple correlation of the increased acetylcholine sensitivity and the fibrillation.

It has been suggested that the increased membrane sensitivity and possibly the initial phase of activity in the muscle nuclei are due to toxic products of the disintegrating nerve.[13] However, it seems improbable that the influence of such products would involve the whole length of a muscle fibre and yet not affect adjacent non-denervated fibres. The multiple innervation of the frog's sartorius[6] has enabled this hypothesis to be tested. One branch of the motor nerve is cut and at least two weeks are allowed for degeneration. Testing by the local application of acetylcholine shows that, in contrast with totally denervated muscle fibres, there has been little or no increase in sensitivity either at the region of the degenerated motor nerve endings or elsewhere along the muscle; that is, that the characteristic increased sensitivity of degeneration is not seen when the muscle fibres still have one zone of intact innervation. Thus there is no indication that any toxic products are liberated by the degenerating nerve endings.

The increased sensitivity characteristic of the whole length of the degenerated muscle membrane occurs only when there is lack of a living nerve connexion. Therefore there appears to be a true trophic influence of the nerve on the muscle, an influence of the same general character as that observed within the confines of a single cell. When the nerve regenerates and reestablishes connexion with the muscle, the trophic influence is restored. Detailed study of this restorative process might throw some light on the nature of the trophic influence. Until this is known treatment of degenerating muscle can only be symptomatic.

Though the atrophy of degenerating muscle resembles disuse atrophy, it has recently been suggested that it may be due to overactivity of the muscle resulting from the fibrillation.[15] However, this seems improbable, for the slight tension in the muscle indicates that fibrillation is due to only a slight activity of the muscle—only a small fraction of the muscle fibres can be fibrillating at any one time. This view seems finally to be excluded by Solandt and Magladery,[11] who found that denervated muscles still atrophied when fibrillation was prevented by administration of quinine.

The contrary hypothesis is that, despite the fibrillation, atrophy is due to underactivity, that is, it is a disuse atrophy. This encounters the difficulty that almost all attempts to prevent the atrophy by directly stimulating the muscle have been practically ineffectual.[7,8,3] However, Fischer,[3] in experiments on rats, found that the stimulated muscle (ten to twenty minutes per day) showed a striking increase in weight compared with the resting denervated muscle of the opposite side. He employed intermittent tetanization and took care to make his stimuli

maximal for the muscle. In all other investigations it appears that the stimuli were so weak that only the superficial muscle fibres were stimulated. Also prolonged direct-current impulses were usually employed instead of tetanic stimulation.[3] In order to mimic natural activity as far as possible the stimuli must be short intermittent series of tetani, such as used by Fischer,[5] and the disposition of electrodes and strength of stimuli must be such that all muscle fibres are excited. We are at present investigating the effects of various durations of this type of stimulation on degeneration atrophy in the different muscles of the cat's leg.

Apart from the possibility that direct muscle stimulation may have some therapeutic value in muscle degeneration, the best treatment seems to be a few weeks' initial rest and then daily massage and passive movements to aid circulation and prevent fibrous contractures.[3] There is no known method of accelerating to any appreciable extent the growth of nerve fibres from the central stump.[17]

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THE NORMAL APICAL PRECORDIAL LEAD OF THE ELECTROCARDIOGRAM.

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BEFORE 1937 the value of the precordial lead in the composition of the human electrocardiogram was fully recognized and widely utilized; but considerable confusion existed among authoritative cardiographers of all countries, because of the technical multiplicity of the derivations employed. One of the most popular and satisfactory was the so-called "Wolferth" lead in which the right arm electrode was placed upon the cardiac apex, and the left arm electrode upon the left leg. The resulting tracing proved to be capable of furnishing reliable and consistent evidence confirmatory of that presented by the standard leads, besides occasionally, though rarely, demonstrating the only abnormality to be seen. The polarity of the deflections obtained by the "Wolferth' 'derivation, however, was opposite in sign to that of the limb leads. In order to unify the nomenclature and technique of the precordial lead, joint committees of the Cardiac Society of Great Britain and Ireland, and of the American Heart Association[1] recommended that "those who employ a single precordial lead should place the electrode (a circular electrode between 2 c.m. and 3 c.m. in diameter) upon the extreme outer border of the apex beat, as determined by palpation. If the apex beat cannot be located satisfactorily by palpation, the electrode may be placed in the fifth intercostal space just outside the left border of cardiac dullness, or just outside the midclavicular line if percussion of the heart is unsatisfactory. It is recommended that a single præcordial lead in which the præcordial lead has the location specified be known as Lead IV R when it is paired with an electrode on the right arm, and Lead IV F when it is paired with an electrode on the left leg. It is suggested that for all ordinary purposes Lead IV R or Lead IV F be employed. The latter lead should have the preference until it has been established that the former, which is somewhat more convenient, is equivalent to the latter for all practical purposes, or yields results of equal value."

Such an authoritative and welcome request was gladly acceded to by the great majority of cardiographers. Lead IV *F* has replaced the "Wolferth" lead in all cardiograms recorded by the Cardiographic Department, Royal Prince Alfred Hospital, since the middle of 1938. The staff of this department had decided to review their results after one year, but unforeseen circumstances have disrupted this programme. As routine cardiography must continue, with, perhaps, new workers to supervise it, it appeared desirable to record the characters of 50 tracings obtained by lead IV *F*, in 50 wholly normal individuals, preferably of one sex and age, and to compare the outcome with reports of observers overseas (Deeds and Barnes,[2] Shanno,[3] Geiger,[4] Hoskin and Jonescu[5] *et cetera*). The subjects chosen were student nurses attending the preliminary training school of the hospital. They were all healthy girls, of an average age of twenty-two years, who had been carefully surveyed clinically and shown to possess no history nor signs of circulatory derangement. All the tracings were obtained by the same observer, using the "G.E." cardiograph, and all subjects were recumbent. The provisions in regard to the placing of the electrode at the outer border of the apex beat, the size of the electrode and the use of electrode paste, as recommended by the Joint Committee, were carefully followed. The importance of securing a flat base-line was chiefly respected by the use of proper electrode paste and well-charged batteries, tranquillity of the subject, and meticulous standardization.

Results.

The results obtained are summarized in Table I. Figure I graphically represents the average complex.

Although a number of similar reports are already available, few only are strictly comparable with this series, while a number of more comprehensive investigations include records obtained by placing the precordial electrode at a number of fixed positions internal and external to the apex beat. It is therefore not easy for the casual cardiographer to obtain quantitative standards for use in the interpretation of lead IV *F*. The best analysis has recently been published by Deeds and Barnes,[2] who examined 50 normal females and 50 normal males by all orthodox precordial leads. The relevant numerical averages obtained by them in respect of the various deflections constituting lead IV *F* are set out in Table II.

TABLE I.
Comparative Numerical Values of the Components of Lead IV F.

		Deeds and Barnes.	Shanno.	This Series.
Number in series		50	100	50
Average age		22	18–22	22
P waves (in millimetres)	Maximum	+0·6	+1·0	+0·5
	Minimum	−0·2	−1·0	−0·25
	Average	0	0	+0·5
Average *P-R* interval (in seconds)		—	—	0·14
Average *Q* waves (in millimetres)		0	0 (One=0·5)	0 (One=0·5)
R waves (in millimetres)	Maximum	+39·0	+18·0	+14·0
	Minimum	+1·2	+1·0	+1·5
	Average	+7·3	+4·0 to +8·0	+5·6 (R_4 slurred in 5 cases)
S waves (in millimetres)	Maximum	−27·0	−23·0	−13·0
	Minimum	−2·2	0	−0·5
	Average	−14·3	−6·0	−2·5 (S_4 slurred in 2 cases)
Duration of *QRS* (in seconds)	Maximum	—	—	0·1
	Minimum	—	—	0·04
	Average	0·08	—	0·07
RS-T deviation (in millimetres)	Maximum	+2·6	+2·0	+1·0
	Minimum	0	0	0
	Average	+1·0 (in 2 cases: −1)	0	+0·75
T waves (in millimetres)	Maximum	+9·3	+7·0	+5·0
	Minimum	+1·3	+1·0	+1·0
	Average	+5·4	+2·0 to +4·0	+2·0

It will be observed that my results correspond to those of Deeds and Barnes with the following exceptions. The average voltages of *R*, *S* and *T* are greater (approximately double), and upward deviation of the *RS-T* segment is commoner in the American figures, but the same pattern is presented, and my values are within the limits permitted by these authors. Shanno's[1] material was identical with mine (100 student nurses, mainly twenty-two years old). His electrode, however, was placed on the cardiac apex

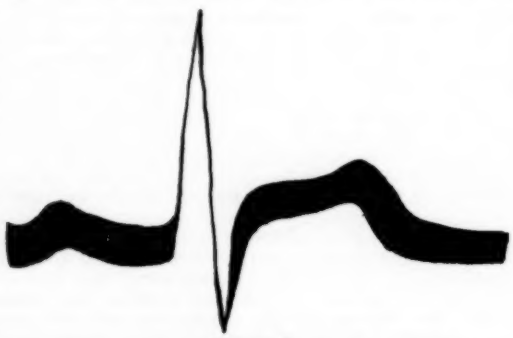

FIGURE I.
Scale pattern of complex of Lead IV *F* compiled from average of values found in 50 normal girls.

fluoroscopically. This refinement, while scientifically preferable, was not recommended by the joint committee referred to, and is too accurate and impracticable for routine hospital or bedside cardiography. This may have been responsible for the slight discrepancy to be seen in Table I. Thus, in one of his subjects a negative *T* wave in lead IV appears, probably because of an unusually wide distance between the clinical and fluoroscopic cardiac apex.

Investigation was made in ten instances of the changes in lead IV *F* which occurred when the electrode was moved one inch, (*a*) to the outer, (*b*) to the inner side of the outer border of the apex beat. It appeared to me to be important to know the extent to which the curve would be thus modified, as such is within the margin of technical error to be expected if cardiography is performed by a lay technician. The results appear in Table II, and make it obvious that no such accuracy as advocated by Shanno is called for in practice. In fact, our complexes were less variable than those described by Shanno.[3] Barnes and Deeds[2] have shown that the chief deflections of this lead (IV *F*) are modified as follows by alterations in the positioning of the precordial electrode along the horizontal plane:

Q becomes more common and pronounced on shift of the electrode to the left.
R becomes higher on shift of the electrode to the left.
S becomes smaller on shift of the electrode to the left.
T becomes smaller on shift of the electrode to the left, but never isoelectric.
RS-T becomes isoelectric or even negative on shift of the electrode to the left.
No change occurs in the *QRS* duration.

My few observations on this point agree (Table II). When the electrode is placed as far medially as in a line midway between the left parasternal line and the left mid-clavicular line, or as far laterally as the left anterior axillary line, the minimum size of the deflections *R* and *T* (that is, those of greatest clinical interest) does not vary a great deal; but a different minimum quantitative standard must be observed in the interpretation. It is none the less important for the technician to approach the outer border of the apex as closely as possible. Posture is important. In comparing tracings of lead IV *F* from

TABLE II.

Average Effect of Shifting Precordial Electrode to Right and Left of Standard Lead IV F Position (Ten Subjects).

—	*Q.* (mm.)	*R.* (mm.)	*S.* (mm.)	*S-T.* (mm.)	*T.* (mm.)	*QRS.* (sec.)
Electrode placed one inch medial to apex beat	—	+4·2	−4·4	+0·3	+3·1	0·07
Standard position (outer border of apex beat)	1=0·5	+5·6	−2·5	+0·75	+2·0	0·07
Electrode placed one inch lateral to apex beat	1=−1·0	+6·5	−3·0	+0·15	+2·8	0·06

TABLE III.

Average Values of Components of Lead IV F in Twelve Male Medical Students after Mild Exercise.

Average Age.	*Q.* (mm.)	*R.* (mm.)	*S.* (mm.)	*S-T.* (mm.)	*T.* (mm.)	*QRS.* (sec.)
20 years	—	+11·3	−8·0	+0·75	+7·4	0·08

different sources, the effect of posture, as shown by Stewart and Bailey,[6] must be eliminated.

Standards for males are not included in this inquiry apart from those mentioned in Table III, which represents some results in healthy male students soon after mild exercise. In so far as the abnormalities of significance in lead IV *F* lie chiefly below the minimum normal values, it is more important to recognize where these values lie as applied to women, who habitually give lower values. Voltages in the male are on the average higher (Deeds and Barnes, Table III).

Analysing the deflections *seriatim*, the following facts emerge:

1. *P* is usually isoelectric, and its form has no clinical significance.

2. The *P-R* interval, where measurable, corresponds to that of other leads.

3. The *Q* wave is almost always absent. It occurred once only in Shanno's series, and once in my own accompanying a *Q* wave in lead I, attaining not more than 0·5 millimetre in each case.

4. None of the huge *T* waves observed by Deeds and Barnes were seen. No importance attaches to the maximum *R* deflexion, but considerable interest must be paid to this deflexion when it is under two millimetres high, and confirmatory evidence of abnormality must be sought elsewhere in the cardiogram. Slurring occurs occasionally in normal tracings, and often appears to synchronize with slurring of *QRS* in lead III.

5. An *S* wave is not necessarily present; but it is rarely completely absent. Its minimal depth is unreliable as a clinical standard of normality. No correspondence to the presence or size of *S* in lead I was noted.

6. The duration of *QRS* does not exceed 0·1 second, but may be slightly longer than found in the limb leads.

7. The *RS-T* deviation is usual and occurs in the absence of digitalis medication. This must be remembered. It takes the form of a gradual and direct upward gradient, averaging one millimetre of positivity, but may reach to 2·5 millimetres. The elevation is generally slightly greater at the distal (or *T*) end than at the commencement (*R* or *S* end). Deeds and Barnes record that a depression may occur rarely in normal tracings. It was not seen by me. Digitalis exaggerates the positivity of *RS-T* and *T* deflexions.

8. *T* waves should be always positive. At least this would appear to be the correct attitude to assume at present. *T* waves less than one millimetre high (that is, isoelectric or slightly negative) should arouse sufficient suspicion to call for caution in accepting the cardiogram as normal, and demand a full clinical and cardiographic review of the situation. Huge *T* waves were not observed, and are not common in *CF* leads, so that it is not possible to express an opinion from these results on the clinical significance of such exaggerated deflexions. A notched *T* wave was seen once by Deeds and Barnes. Evans places the normal value of *T* as three millimetres; but such a standard is not supported by this investigation. No diphasic *T* waves were encountered, although Wolferth and Wood[7] have found diphasic *T* waves in persons "in whom the presence of heart disease could not be proven". No consistent correlation was observed between the *T* waves of lead I and lead IV *F*.

The relative value of lead IV *F* as compared with lead IV *R* was not investigated. *CR* leads appear to give higher minimum values and more level base lines than *CF* leads, and so are preferred by some workers—for example, Deeds and Barnes. We have always managed to secure a steady horizontal base line by the use of a good electrode jelly thoroughly massaged into the skin, the recumbent posture and proper relaxation of the subject, and by maintaining all battery voltages at their full values; and Geiger states that in 55 of 400 tracings abnormalities occurred to a significantly greater degree in lead IV *F* than in lead IV *R*.

Conclusions.

1. Fifty cardiograms obtained from healthy young women were analysed in respect to the precordial lead IV *F*, wherein the exploring electrode is placed on the outer border of the apex beat. Slight inaccuracy in this latter respect was shown to be immaterial.

2. In women a definite *Q* wave, an *R* wave under 1·5 millimetres, a *T* wave of under 1·0 millimetre and a positive deviation of the *RS-T* of more than 2·0 millimetres are abnormalities which should call for careful revision of the appearance of the limb leads in conjunction with a full review of the clinical facts.

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RIGHT-SIDED VISCEROPTOSIS.

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DURING the past nine years, in the private practice of the Brisbane Clinic, 541 case records have been classified under the disease group "abdominal visceroptosis". This is a general term which includes at least the following classes of cases.

The greater number present certain readily recognizable syndromes which we believe are associated with non-fixation and prolapse of the ascending colon, and this finding has often been confirmed radiologically or at operation.

A second group are basically examples of abdominal neurosis; but because the symptoms have a postural pattern, are included under this heading rather than with the large group labelled "neurosis".

A third group presents an indefinite clinical story, which does not allow of their relegation to other classes of abdominal lesions; but because of the radiological findings they are included, sometimes erroneously, in this group.

In a small number of cases perusal of the records fails to show the reason for such classification; but incompleteness of the written record rather than error on the clinician's part may be the usual explanation of this group.

The frequency of the diagnosis can be gauged from Table I, which enumerates the case records in the abdominal classifications.

TABLE I.
Abdominal Complaints.

Diagnosis.	Number of Cases.
Colitis	48
Chronic duodenal ileus	16
Appendicitis	78
Duodenal ulcer (verified radiologically)	262
Gastric ulcer (verified radiologically)	68
Gall-bladder disease	321
Hernia	112
Neoplasms	104
Visceroptosis	541
Nervous dyspepsia (abdominal neurosis)	468
Incompletely verified	1,536
Total	3,554

If the incompletely verified cases are excluded, the visceroptotic group is seen to be larger than any other single group of abdominal complaints.

Age and Sex Groups.

The commonly expressed opinion, regarded as axiomatic by earlier writers, and confirmed statistically by Carslaw[1] that visceroptosis is much more frequent in women than in men (women : men = 3·25 : 1), is not borne out by these records, for of the 512 patients whose records were analysed, 267 were females and 245 males.

The grouping at consultation by ages in decades is as follows:

10 to 20 years	4·5%
20 to 30 years	33·2%
30 to 40 years	31·4%
40 to 50 years	22·0%
50 to 60 years	8·6%
60 years and over	0·3%

As in the practice under review few children are treated, these age groups do not form a parallel to the distribution of the complaint in the general population; but for the age groups above adolescence they should be reasonably accurate.

Carslaw published a very similar table, confirming the fact that patients presenting the disorders under discussion seek medical advice during their active working years, most often between the ages of twenty and forty years.

Previous Abdominal Operations.

In the compilation of records a routine note is made of previous illnesses or operations, and a study of the written histories has often enabled the success or otherwise of such operations to be established.

Of the 512 patients, no less than 173 had submitted to previous surgical procedures, much more commonly the women than the men, for while 104 (or 40%) of the female patients had surgical abdominal scars, only 69 (or 28%) of the men had been the subjects of previous abdominal operations.

In 138 cases the operation was thought to have been appendicectomy, and in 69 of these the history stated that no lasting benefit was obtained. In the remaining 69, the nature of the present symptoms and the time relation of the operation to these symptoms often created the presumption that in their cases, too, the appendicectomy had been equally unsuccessful.

Of the remaining operations, eleven had been operations on the gall-bladder and four were noted as unsuccessful; eight were hysterectomies; while there were many lower abdominal sections in females for such variously described entities as cystic ovary, inflamed tubes and ovaries, retroversion of the uterus *et cetera*.

The greater number of unsuccessful operations performed on this group of patients is a sad commentary on the diagnostic ability, and indirectly on the medical education, of a large number of practitioners.

The teachings of a past generation of surgeons concerning such a nebulous entity as "chronic appendicitis" are being displaced with such dangerous slowness that one must feel very dubious whether the present voluntary methods of post-graduate education do not need drastic revision in order to force modern truths (that may conflict with its undergraduate teaching) upon the practising profession.

To those of us who see many referred patients, it is evident that the professional tendency in the presence of chronic right lower abdominal pain and tenderness is still to act as though such pain were always due to the appendix, rather than to accept the real truth that such a diagnosis is almost the last explanation to consider.

The Syndromes of Right-Sided Visceroptosis.

Analysis of the histories elicited from these 512 patients will show that certain symptoms and symptom complexes repeatedly occur, and the majority of the syndromes elicited are capable of tabulation as follows:

Pain in the right iliac fossa	253 patients
Pain in the epigastrium	109 patients
Epigastric fullness or flatulence	91 patients
Dyspeptic symptoms	78 patients
Relation of symptoms to posture—that is, relief by rest	265 patients
Relation to exertion or bodily movements	235 patients
Constipation	91 patients
Neurosis or emotional instability	178 patients

From this tabulation it becomes clear that the common complaint of these patients was of an abdominal pain or discomfort having a relationship to posture and to bodily movements, and rather often associated with emotional instability or neurosis. This constitutes the clinical syndrome of right visceroptosis; but several subgroups can be recognized in this larger group, and of these the syndrome of the right iliac fossa forms one of the most definite and unvarying in abdominal diagnosis.

From many hundreds of patients (remembering that the group under discussion were seen in private practice and very large numbers of similar patients have been treated in public hospital practice) a story such as this can be obtained. A young adult complains that he, or more often she, has for a long time had a "pain" in the right side. In working girls it often started within a few months of leaving school and of taking up of some factory employment, such as working a power sewing machine; while in girls of higher social status the discomfort began within a year or two of leaving school at the age of seventeen or eighteen years. Though it may primarily be described as "terrible", questioning reveals that the pain is not really terrible, hardly even severe; but its importance is enhanced

either by its constancy or by the implication of serious underlying disease. It is described variously as aching, stabbing, burning or dragging, and its distribution, which is usually limited to the right iliac or inguinal regions, may overflow into the area on the anterior part of the upper portion of the thigh supplied by branches of the first lumbar nerve root. The pain may occur almost daily, or there may be partial or complete relief between exacerbations of discomfort.

Though the pain may be present when the patient lies down, and may occasionally persist in certain horizontal positions, especially the supine position with extended thighs, and sometimes when the patient lies on the left side, it is always possible to find a horizontal posture which will relieve the pain within a reasonable time. The pain is rarely present when the patient is awakened in the morning, but comes on again at a variable time after the assumption of the erect posture. Usually this is after a number of hours, perhaps late in the morning, and the discomfort then persists so that almost always the patient is more uncomfortable late in the day than early. Often the onset of pain can be related to the development of physical tiredness. Cross-examination usually reveals that certain bodily movements or variants of the completely erect posture accentuate the pain, and when the patient is standing, it is particularly noted that the forward sag of tiredness, and bending forward and lifting movements cause this effect. Conversely some men note that if pain is present, a conscious straightening up and bracing back of the shoulders or supporting the lower part of the abdomen with the hands will remove it. The working girl has more discomfort while sweeping than while scrubbing, while pain in the sitting position is associated with bending forward over the power machine, or sagging into the canvas lounge seats at the evening picture show. Riding along a rough road in a motor car and the jolting associated with tractor driving are potent producers of discomfort.

On physical examination, if there has been a recent attack of pain, a localized spot of tenderness may be present at the outer edge of the right *rectus abdominis* muscle about the so-called McBurney's point, the caecum can be displaced by palpation far medially, and radiologically the non-fixation of the ascending colon, with prolapse of the caecum in the erect posture, is confirmed.

The variability of the syndrome concerns, first, the constancy of the pain, which in some cases may disappear wholly or partly for long intervals, to recur in attacks; in the nature and the intensity of the pain; in the time interval between the adoption of the posture and the appearance or disappearance of the discomfort; and in the presence or absence of accentuation by certain bodily movements, especially stooping or lifting. The constant feature is the recurrence of right lower abdominal pain with a postural pattern.

The next most frequent syndrome is that associated with epigastric pain. This may occur alone, or often is the more obvious component of a multiple discomfort involving also some right iliac symptoms. The pain may have the character of an intrinsic gut pain, an ill-defined stomach ache centred on the mid-line; but usually it is an abdominal wall neuralgia, either bilateral or more marked on one side. The postural relationships have the same character and variations as those associated with the iliac fossa syndrome.

The dyspeptic syndromes in visceroptosis are less well defined than the pain syndromes. The character of the dyspepsia varies according to the physical and gastric habitus of the patient, and of itself has no diagnostic value. It is not well appreciated yet by the general profession that the time relationship existing between food intake and pain development does not vary with the origin of the discomfort, but is constant for each individual. It is in effect a part of his general characteristics, and as unvarying as his general build, and the motor and secretory functions of his stomach. In a general way the broad-chested type with tonic high stomach develops pain later after food, whether the stimulus is local, due to ulcer or cancer, or more distant, due to anxiety factors or visceroptosis. Every individual possesses his own brand of dyspepsia, potential or actual, which has therefore no importance in the differential diagnosis.

The feature that draws attention to the visceroptotic origin of a dyspepsia is its postural relationships. These patients state that if only they could live their lives in bed they would have a perfect digestion; but since this is impossible they experience discomfort after certain meals, more commonly those later in the day than earlier, and worse after a tiring day than otherwise. Such clinical pointers, reinforced by an absence of radiological evidence of the other physical dyspepsia-producing factors, and the presence of non-fixation of the ascending colon with prolapse of the caecum in the erect posture, make a strong diagnostic triad.

Earlier writers on the diagnosis of right-sided visceroptosis have described further symptom groups in this disease, and particularly the gall-bladder syndrome and that of chronic duodenal ileus. Relying as they did on factors of physical "drag" producing direct mechanical effects on upper abdominal organs with subsequent stasis, these authors felt able to distinguish such secondary effects on gall-bladder, duodenum and stomach, and separate their clinical syndromes. As will be shown later, these explanations ignored, amongst other difficulties, the peculiar characteristics of visceral pain, and modern knowledge would render their explanations of such pain production infeasible.

Though in past years the authors sometimes felt that they could distinguish a syndrome produced by biliary tract dysfunction, more recent examples of upper abdominal pain in visceroptotics seemed better regarded in the light of unusually high abdominal wall neuralgias.

The chronic duodenal ileus group of right-sided visceroptosis is a purely radiological subdivision. Associated with a greater or lesser degree of gastric retention and dilatation is usually found a large duodenal cap. The meal then pours into the third part of the duodenum, where an irritable focus obviously exists. The meal tends to be held up here for prolonged periods, and dilatation of this portion of the gut is present; but particularly one notes that frequent active retroperistaltic waves are initiated, which force part of the meal back towards and into the duodenal cap.

This focal irritability is much the most characteristic feature of chronic duodenal ileus, and it is the group of cases in which this feature is very pronounced, and in which some stasis in the distal portion of the duodenum exists, to which this term is applied.

Clinically, this group forms part of the dyspeptic group, and in our series we have had no clinical findings which would enable a purely symptomatic diagnosis of such a condition to be made. We have not seen a single case in association with right visceroptosis in which mechanical factors of mesenteric pedicle pressure seemed to be the explanation of the stasis.

The Concept of Right Visceroptosis.

In recent years the tendency to deny any clinical importance to abnormal mobility of the ascending colon and caecum has been so widespread, and such meagre attention, and that often hostile, has been paid to this subject in authoritative text-books, that younger members of the profession may not realize that large numbers of papers, often by very eminent members of our profession, were published on this subject between 1910 and 1930.

The explanations of this recent lack of interest are certainly multiple. The accurate pre-operative diagnosis of abdominal abnormality now possible has limited greatly the necessity for wide surgical exploration, and the pioneer interest in noting and recognizing variations from the normal has abated. The radiologist has become the arbiter of visceral anatomy in the living, and his realization of the wide variations in the normal as regards mobility and position of viscera has led him to accept all degrees of right colon mobility as being a normal and not a symptom-producing finding.

Increasing knowledge of visceral physiology and of its peculiar sensitivity has rendered untenable the older

explanations of symptom production in right-sided visceroptosis, while the complete failure of surgeons in past years to recognize the influence of emotional factors in the production of abdominal symptoms, or in the accentuation of such symptoms, has often led modern physicians to sweep away the physical conception of visceroptosis as wholly baseless.

And finally, from personal observation, one has noted that in North America, at present the leading home of surgical research, young men seem interested only in problems susceptible of proof by laboratory test or by animal experiment. The purely clinical problem, such as is presented here, seems to have for them very little attraction.

Although the recognition of *caecum mobile* as a symptom-producing entity was made by Wilms[2] in 1910, and caecopexy was advocated for its relief, the interest of British surgeons became focused on the subject only after the publication by Waugh[3] in 1920 of an article in *The British Journal of Surgery*, in which he recognized congenital absence of fixation of the ascending colon as the primary factor in symptom production. The common syndromes were described, and purely mechanical explanations were given for their production.

The absence of a lateral purchase for the peristaltic force was supposed to dissipate part of its propulsive power in moving the colon as well as its contents, and stasis in the caecum was thereby induced. The loaded bowel by its mechanical drag through its mesentery or through abnormal membranes produced the varied secondary effects which manifested themselves as the described symptoms.

Interest in this subject was attested in the following years by articles written, amongst others, by H. M. W. Gray,[4] Coffey,[5] McConnell and Hardman,[6] Walton,[7] and Wilkie,[8] the substance of which was crystallized in a paper covering 60 pages of *The British Journal of Surgery* in 1928 by Carslaw,[1] of Glasgow. Though less obsessed than Waugh by mechanistic explanations, he still regarded caecal stasis and dilatation as an essential feature in symptom production.

Many children are undoubtedly born with developmental membranes and a considerable degree of congenital ptosis who do not develop any stasis. Absence of symptoms in spite of these conditions of ptosis is due to the fact that there has been no derangement of the mechanism of peristalsis. The viscera have retained their functional activity, the stomach, though low, is orthotonic, the caecum is not dilated though in the pelvis.

Carslaw noted with approval, however, the views of Tyrrell Gray[9] in the following paragraphs:

Tyrrell Gray agrees with Waugh that the ascending colon is normally sessile, and being supported by its attachment to the posterior abdominal wall, does not exert any traction on its mesocolon, in which lie the vessels and nerves which control its nutrition and movements. If, however, the strain is transferred to the mesentery, there is a mechanical stimulation of the sympathetic nerves, with inhibition and derangement of the normal intestinal movements.

Tyrrell Gray suggests that in right-sided visceroptosis the initiation of symptoms is due to a relative increase in mesenteric tension, the result of either a loss of support or an abnormal loading of the bowel.

Nowhere in this long paper does Carslaw suggest that variability in pain intensity may be due not to variability in the primary stimulus but to changes in the nervous sensibility due to emotional factors.

During the past ten years many surgical text-books in their discussion of right iliac pain have stated briefly that such pain is often due to visceroptosis, but there has been little tendency to stress this as an important factor in the differential diagnosis.

Mechanism of Pain Production in Abdomen and Abdominal Wall.

It seems axiomatic that diagnosis of abdominal disorders will remain largely empirical till an exact knowledge prevails of the mechanism of abdominal pain production, and till that is forthcoming each of us must adopt the theory that best fits his personal experience in this field.

In our view abdominal pain can be produced in the following four ways:

1. There is an intrinsic gut pain expressing itself in the case of the intestinal tract, and its diverticulæ, as an ill-localized discomfort centred on the middle line at varying levels according to the segment of gut involved. This may vary in intensity from a feeling of mild fullness to a severe colic. It is the primary pain of acute appendicitis, the pain of intestinal and biliary colic and gut obstruction, and is produced in the gut wall or perhaps in the adjacent mesentery by a gross derangement of smooth muscle tension. It is produced by stimulation of sympathetic end-organs and passes through sensory autonomic tracts to consciousness. In our experience it seems the only type of pain produced by smooth muscle tension in the abdomen.

2. An inflammatory pain or soreness is felt in the abdominal wall at the site of a primary inflammation, or more commonly when adjacent inflammation of intra-abdominal organs spreads to the abdominal wall. It is a well-defined, accurately localizable, somatic pain similar in all respects to inflammatory pain elsewhere in regions supplied by non-autonomic nerves, except perhaps that the discomfort due to inflammation of parietal peritoneum may sometimes be referred to its corresponding skin segment, and not felt directly at the seat of peritoneal inflammation (Morley[10]). This seems the most feasible explanation of shoulder-tip pain due to diaphragmatic inflammation, but repeated observations (made under local anæsthesia) fail to confirm it over the anterior abdominal wall.

3. Stimulation of unanæsthetized parietal peritoneum, as for instance by rubbing with a gloved finger, causes severe pain, recalling the dictum of Morley that the sensitiveness of this structure is akin to that of the cornea. Such discomfort is elicited to a varying degree by free intraabdominal gas, fluids and blood, also apparently in a mild degree by gross colonic distension (felt over the affected segment and distinct from intrinsic obstructive pain) and even by intense spasm (left iliac discomfort of a spastic sigmoid colon). This pain is also localized accurately to the site of stimulation.

4. Neuralgia of the abdominal wall is the fourth cause of abdominal pain. The sensory nerve supply to segments of the abdominal wall becomes so sensitized that normal stimuli become accentuated during their passage through these nerves and the brain interprets them as arising from much greater stimuli. In this way both spontaneous pain and localized tenderness are produced. Such neuralgic pain is recognized by its character (aching, stabbing, throbbing, burning), its distribution in anatomical nerve root areas, its non-association with inflammatory causes, and the associated history and physical signs. It can be produced by three mechanisms: (*a*) as a local manifestation or part of a general expression of nervous hyperæsthesia (neurosis); (*b*) by direct nerve stimulation; (*c*) as an expression of a sympathetico-somatic reflex. Such a reflex might theoretically be produced by any form of sympathetic stimulation; but as regards the right lower quadrant of the abdominal wall, experience suggests that such pain is never produced by gut inflammation or by smooth muscle tension, is infrequently produced by experimental stimulation of the gut mesentery, but, particularly in circumstances of increased nervous awareness, is frequently elicited as the result of mesenteric drag.

If these theories of abdominal pain are applied to the symptoms under discussion in this paper, it will be obvious that the lateral pains are to be regarded as abdominal wall neuralgias exclusively, while the epigastric discomforts, according to their character, may represent either intrinsic gut pain or bilateral wall neuralgia. Such mechanisms as caecal distension or partial volvulus, involving smooth muscle stimuli (postulated by Carslaw) are seen as invalid explanations of localized lateral pains.

The Nervous Factor in Right Visceroptosis.

In the case histories of 178 patients it will be noted that neurosis or emotional instability has been remarked. The relationship of these nervous states to the syndromes under discussion has been much discussed, and many

physicians have thought that the symptoms are actually of neurotic production and the postural pattern assumed in this particular group is largely fortuitous.

The suggestion that the neurotic factor is primary cannot be accepted by the experienced clinician as generally true, although, as was mentioned in the introductory paragraphs of this paper, some cases classified by us as of visceroptosis fall into this category.

Observation of many adolescent visceroptotics, particularly in out-patient practice, has convinced us that in the early stages of symptom production a neurotic factor is not usually present; but this factor becomes more noticeable as the length of history increases.

In the majority of cases neurosis seems the result of years of chronic pain; the neurotic symptoms are secondary to the mechanically produced discomforts. In this regard two nervous mechanisms seem particularly important, the first the role of anxiety in the production of symptoms, and the second the role of continued sensory stimulation.

The place of anxiety in the production of abdominal symptoms cannot be over-estimated, and there are few subjects of chronic illness in whom such a factor is not a partial explanation of their state. Although we profess no special knowledge of psycho-somatic pain, some simple explanation like the following seems to clarify the clinical problem. Anxieties of external origin or due to supposed bodily departures from the normal (for example, the general symptoms associated with irregular defæcation) tend to cause, beside a general nervous tenseness, less or greater according to the inherited stability or otherwise of the individual, more limited discomforts, usually due to localized hyperæsthesias. Like a volcanic force attempting to escape through weak spots in its crust, anxiety expresses itself by a fixation on areas of physical inferiority, or where physical disease is believed to be common or serious. Thus in numberless cases in which no underlying disease is present, anxiety expresses itself as a neuralgia in the right lower quadrant of the abdominal wall, because the patient believes this is an area of common or dangerous diseases. Associated with this spontaneous pain is the tenderness at McBurney's point, where the sensitive eleventh intercostal nerve at the outer edge of the *rectus abdominis* muscle is most exposed to palpation. Almost equally as frequent as the iliac fixations are the neuralgias of the left wall of the chest, the region of "heart disease".

Where physically produced discomfort already axists, as in the visceroptotic, the accession of anxiety symptoms accentuates the discomfort, while at the same time diminishing the accurate postural relationship existing in the pure physical syndrome. Quite often the anxiety of the visceroptotic is due entirely to uncertainty as to the cause of his physically produced trouble, and the same pain, incapacitating when regarded as due to the potentially dangerous "chronic appendicitis", is acquiesced in cheerfully when only due to a "sagging colon" or to post-operative "adhesions".

Parenthetically it may be said that the character and distribution of the pain in the chest wall of most women with "chronic mastitis" have similarly all the characteristics of a nervous fixation of an anxiety state.

Within the sensory areas of the autonomic nervous system stimuli not commonly rising to the level of consciousness may become actual discomforts as the result of this increased awareness, a common example being the appreciation of gastric filling as a discomfort ("fullness" or "flatulence").

The physiologist can demonstrate by a simple experiment that the repeated passage of an impulse along a nerve produces facilitation in that nerve so that subsequent stimuli become ever more capable of eliciting a response.

Whether recurrent sympathetic stimulation is more likely than somatic stimulation to produce neurosis is an interesting speculation; but whether or not this is so, a recurrent sympathetico-somatic reflex will become easier and easier of repetition, while also the effects of anxiety will increase the sensitivity of either or both of the sensory components concerned.

The Production of Symptoms in Right-Sided Visceroptosis.

There is a congenital failure, partial or complete, in the usual lateral (or dorsal) fixation of the ascending colon in about 20% of persons. Many of these never, and none of them at all times, experience discomfort, and the explanation of this intermittency should provide the key to the treatment of such troubles when they arise.

It is our belief that the production of symptoms in right-sided visceroptosis is associated with three distinct factors: (i) non-fixation of the ascending colon, (ii) a failure of secondary postural supports, (iii) a nervous facilitation.

Always two and often all these factors must act before the production of serious symptoms occurs.

Non-Fixation of the Ascending Colon.

In the Australian Institute of Anatomy at Canberra, Sir Colin MacKenzie[11] collected a magnificent series of comparative specimens which show how the gradual assumption of the erect posture has necessitated a measure of dorsal fixation in the colon. Beginning earlier in the left side of the colon, it has here become so well established that in *Homo sapiens* mobility of the descending colon is a rare abnormality. Fixation of the right side of the colon, however, is much more experimental and unstable, and even in man a congenital defect in fixation is present at birth in about one baby in five. This fixation starts at a mesial fixation area below the pylorus and from here proceeds downwards, fixing the mesentery to the duodenum and the dorsal wall. Defects of fixation are therefore more common in the ileo-caecal region than towards the hepatic flexure.

The varieties and degrees of this abnormal mobility have been considered exhaustively by McConnell and Hardman and by Carslaw, and it is necessary only to restate here that while usually the ascending colon has become a retroperitoneal organ by fusion and obliteration of its mesentery, in 20% of individuals this mesentery persists in whole or in part. In the erect posture the full weight of the right side of the colon is then distributed along the narrow line of the posterior attachment of the mesentery, instead of being disposed over a broad area sideways, as when no mesentery is present. In the supine position the caecum can be palpated medially till the right iliac fossa feels empty, while radiologically a wide variation of position can be achieved with variation in posture and by palpation. In the erect position the hepatic flexure is often below the line of the iliac crests, while the caecum may be in the true pelvis.

Secondary Postural Supports.

Secondary postural supports have been discussed by one of us[12] in a previous communication. In the absence of the usual lateral fixation of the ascending colon, the factors that hinder free prolapse of the caecum into the true pelvis are, first, the primitive dorsal mesentery, the continuation of the ileal mesentery to the caecum, and secondly, and more important, the existence of an abdomino-pelvic isthmus (Figure I).

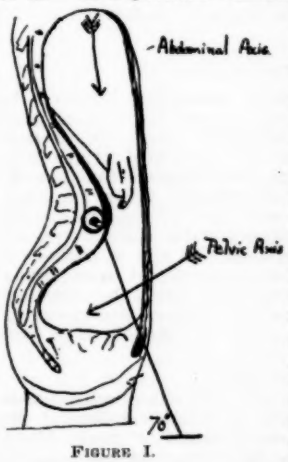

FIGURE I.

The obliquity of the pelvic brim is normally such that it maintains in the vertical position an angle of 70° with the horizontal. Not only does this cause definite constriction to exist between the two cavities, but also the axes of these two cavities are actually flexed on each other. A flat abdominal wall, inasmuch as it forms one side of this isthmus, contributes in an important way to its narrowing and *vice versa*. Sudden

jolts or jars and the increased abdominal pressure resulting from straining at physical work are factors which help to facilitate colonic descent. The abdominal and pelvic cavities are pivoted on each other at the lumbo-sacral articulation, and increase of their angulation to each other increases the dorso-lumbar concavity of the back (producing lordosis), while decrease of their angulation decreases or undoes lordosis.

Factors, then, that widen and straighten the abdomino-pelvic isthmus are: (i) a decrease in lumbar lordosis, (ii) lack of tone in the anterior abdominal wall, (iii) stooping, which tends to convert the abdominal and pelvic cavities into one straight tube.

Now we have already seen that these are exactly the postural factors that favour the production of symptoms in right visceroptosis. It can be stated with a considerable degree of certainty: (i) that a right visceroptotic has symptoms when the caecum prolapses towards the true pelvis; (ii) that such prolapse occurs only when certain secondary factors of posture and muscle tone are brought into action; (iii) that the secondary factors act by (*a*) widening and reducing the angulation of the abdomino-pelvic isthmus, (*b*) increasing the weight of the caecum, (*c*) facilitating its descent by jarring and jolting and by increasing intraabdominal pressure.

These secondary postural supports tend to become inadequate, especially in the tall, thin type of person. Though the incidence of lack of right colon fixation is presumably constant in any group of babies, those who grow tall and thin are especially apt to develop symptoms, as the analysis in Table II shows.

TABLE II.
Visceroptotics.

Height.	Number.	Average Weight.	Normal Average Weight for Height.
ft. in.		st. lb.	st. lb.
5 2	3	8 5	9 0
5 3	2	8 4½	9 7
5 4	9	8 10	9 3
5 5	4	9 11	10 5
5 6	16	9 2½	10 7
5 7	13	10 0	10 8
5 8	32	10 4	11 1
5 9	28	10 8	11 8
5 10	35	10 5	12 1
5 11	16	10 13	12 6
6 0	14	11 9	12 10
6 1	8	10 7	13 0
6 2	1	9 10	
6 3	1	10 7	

Such a table possesses additional significance in showing the importance of a purely physical factor in the production of symptoms in these patients, as opposed to the neurosis school of thought.

Nervous Facilitation.

Even should the secondary supporting factors be inadequate, and prolapse of the caecum occur, it is probable that the resulting mesenteric drag is usually symptomless. In order that the postulated sympathetic stimulation may derange gastro-intestinal function by inhibition or by overflow into somatic nerve segments, a facilitation of the impulse either by frequent recurrence or by the intermediation of anxiety states seems necessary. In this way only can the variability in age of commencement and the capriciousness of the symptoms be explained. It is shown also by the frequency with which the symptoms can be temporarily abolished by nervous sedatives not possessing anodyne action, such as phenobarbital and bromides.

Summary.

To summarize, we believe that the older writers were wrong in assuming that with abnormal mobility of the ascending colon symptoms were produced by caecal dilatation or by volvulus. We do not believe caecal stasis is synonymous with constipation, or in fact that such stasis is often present.

In particular, we do not believe that a chronic gastro-intestinal stasis not resulting in abnormal fluid or electrolyte losses would produce general toxic effects, but rather that such general symptoms when present are due to an anxiety state.

We believe that the discomforts of right visceroptosis are produced by a sympathetic sensory stimulation, initiated in the caecal mesentery, in response to factors already defined, and that symptoms are produced either by a gastro-intestinal derangement due to sympathetic inhibition, or as an intercostal neuralgia due to overflow of the autonomic stimulation into sensory somatic nerves.

Differential Diagnosis.

As regards the right iliac fossa syndrome of visceroptosis, if the explanations advanced here are to be rejected, some other rational cause must be adduced. In historical sequence the alternative explanations may be tabulated as: (i) chronic appendicitis; (ii) orthopædic disorders of the back, and especially lordosis; (iii) colonic dysfunction, or constipation; (iv) urological states; (v) abdominal neurosis.

Chronic Appendicitis.

The view that chronic pain in the right iliac fossa with the postural characteristics already described could be due to "chronic appendicitis" should now be a matter of historical interest only and would hardly be worth discussion were it not that treatment is often still based on such a view (and this does not apply only to the medical outposts). It belongs to an era when the appendix was invoked as the cause of any ill-understood abdominal state and when no distinction had been drawn between visceral and somatic sensation.

With such limited knowledge, localized tenderness suggested underlying inflammation; the appendix underlay this area and was believed to be a common source of inflammation; the pathologist found changes in the appendix such as might follow chronic inflammation; what more was necessary? That the symptoms usually recurred rapidly was not always brought to the same surgeon's attention, or if it was, he assumed that the recurrence of symptoms was due to abdominal adhesions which were an unfortunate frequent sequel of abdominal surgery.

The fact that removal of the appendix sometimes relieves chronic iliac pain shows only that the removal of anxiety may depress stimuli again below the level of consciousness. How little importance the modern surgeon attaches to the appendix as an explanation of chronic discomfort (of any sort) may be gauged from the fact that during a recent period of five weeks spent by one of us in following the surgical practice of two American group clinics, where many thousands of chronic abdominal ailments are treated yearly, only one appendix was seen removed (and this was the sequel of an appendiceal abscess, an active duodenal ulcer being also present and causing the symptoms necessitating operation). In our own series of 3,554 patients with abdominal complaints, only 78 were classified as suffering from appendicitis.

Orthopædic Disorders of the Back; Lordosis.

During many years the view of Goldthwaite[12] and the Boston school have been kept constantly in mind during the examination of patients with the right iliac syndrome. With regret we must state that the simple explanation of intercostal neuralgias adduced by this school will not work. Most of these patients show no back defects, in either the standing or sitting position, and no lordosis. The initiation of symptom-production is by a forward flexion, an undoing of lordosis; while the sway-back lordotic type of patients possess the angulated abdomino-pelvic isthmus that provides an effective secondary support for the mobile caecum. The constant association of a loose ascending colon with this syndrome is inexplicable on an orthopædic basis.

Colonic Dysfunction or Constipation.

In many quarters where the concept of right-sided visceroptosis is unknown or not accepted, the relation of

right iliac pain to the colon is still accepted. A loaded colon, a spastic caecum, or a nervous colitis are terms applied to the supposed underlying condition. Any explanation which regards a disturbance of colonic function as a cause of lateral pain takes no account of facts concerning the site of visceral pain, or else calls in a theory of referred pain from smooth muscle stimulation which experience rejects as non-factual. It also fails to explain the constant association of non-fixation of the ascending colon, or the variability induced by posture.

Urological Explanations.

Another school of thought, while refusing to accept the ptosis explanation for right iliac pain, attempts to incriminate the urinary system. Though occasional secondary effects of caecal ptosis implicate the right kidney, we state confidently that in the great majority of these cases the most painstaking investigation reveals no abnormality in the right kidney or ureter.

Any individual surgeon has the duty to convince himself by adequate urological investigation of this fact; but after that the routine making of pyelograms in every case of chronic right-sided pain becomes a deplorable and uneconomical example of machine diagnosis.

The establishment of the upper abdominal syndromes of visceroptosis has already been discussed. Very careful history-taking is of the first importance, and as with the other dyspepsia-producing entities, the ultimate diagnosis rests usually on an integration of the clinical and radiological findings.

The Treatment of Right-Sided Visceroptosis.

If the thesis is accepted that the production of symptoms in right-sided visceroptosis is the resultant of three factors—(i) congenital absence of lateral fixation of the ascending colon, (ii) failure of secondary supporting factors, and (iii) nervous facilitation—it seems obvious that the factors whose variation precipitates discomforts should be corrected before any attempt is made to correct the congenital factor.

To relieve the anxiety which so frequently determines nervous factors, explanation and reassurance are of first importance. Besides being an important diagnostic procedure, X-ray examination after a barium enema is a most important therapeutic measure, especially when neurotic fixations are present. The patient must be made to realize that the physician understands his condition and that everything necessary has been done to exclude other possible causes. He must be told specifically that he is not suffering from an ulcer or a cancer or from chronic appendicitis, and that his discomforts arise from no underlying conditions that need shorten his life or impair his efficiency. Before the patient leaves the consulting rooms, the physician should be quite sure that he has a full understanding of his condition and is quite satisfied with the explanation.

This education is a most important part of the treatment. The intelligent patient will usually agree that his condition has been correctly described, and very often will state that he is now perfectly satisfied. It was not the intensity of the discomfort that worried him, but its implications of underlying dangers, and, once reassured, he is willing to acquiesce in any discomforts entailed by Nature's failure to perfect her job on him. A bromide mixture or phenobarbital is usually prescribed, to be taken for a brief interval and then kept for use at times of future discomfort. It is quite rare for these measures, during their administration, not to result in a complete remission of symptoms.

For those who still complain of symptoms one endeavours to be sure that the patient is satisfied that the diagnosis is correct and the mechanism understood. A visceroptosis corset belt may be ordered. These usually give relief by upward support and probably by increasing intraabdominal tension.

At the same time efforts should be made to reestablish the supporting postural factors. This, we feel sure, is less likely to be achieved by physiotherapy or by gymnastic exercises than by the rules laid down in such a book as "Exercise without Exercises".[10] This author emphasizes that all these gymnastic health measures are designed towards the production of a normal bodily posture. Though muscle development may be valuable in itself, the production of a good posture will be attained only when the requirements embodied in the injunctions "sit tall", "stand tall" and "walk tall", are consciously applied so frequently that they become automatic. When such a posture is acquired, the abdomino-pelvic isthmus is reestablished and will fail only when forward flexion of the abdominal axis is combined with an increase of intra-abdominal pressure.

In such circumstances prolapse of the mobile caecum must occur, and if the drag rises to consciousness, either the resulting discomfort must be acquiesced in or measures must be taken to fix the ascending colon surgically. Since only 29 out of 512 patients were submitted to colopexy, it is obvious that the necessity for this surgical fixation is thought by us to be rarely present. It should never be undertaken till the medical measures outlined have been given a prolonged trial, and even if discomfort remains, efforts should be made to induce the patient to acquiesce in the continuance of such discomforts.

A small minority of patients are left, the continuance of whose livelihood depends on removal of their pain, or in whom the development of severe neurosis from the chronic discomfort seems inevitable, and in this group colopexy should be considered.

Colopexy and its Results.

When surgeons who have made gastro-jejunostomy their routine operation for chronic duodenal ulcer or who have frequently performed cholecystectomy for non-calculous gall-bladder syndromes, criticize the operation of colopexy on the grounds of its results, they are skating on very thin ice. Both published statistics and personal experience show that when the operation is properly performed in selected cases the majority of patients experience a gratifying relief from previous symptoms.

The usual success of non-surgical measures will probably make the accumulation of large modern series of colopexies impossible; but in earlier years some impressive statistics were collected. Carslaw agreed to operate on every patient brought to him whose condition was, in his opinion, primarily due to abnormal mobility and prolapse of the ascending colon, no matter how far advanced were the secondary results. Two hundred and forty-two operations were performed between 1920 and 1926, and in 1928 the condition of 239 of the patients was ascertained. Two hundred and thirteen were satisfied with the result of the operation, while of 51 who had undergone previous operations without benefit, a right colopexy cured all but eight.

TABLE III.
Results of Colopexy according to the Type of Case.

Type of Case.	Cured.	Much Improved.	Failed.	Total.
Gastric type	27	9	5	41
Duodenal type	21	8	3	32
Right iliac fossa type ..	86	17	4	107
Renal type	5	1	0	6
With gastric or duodenal ulceration	15	2	8	25
With chronic duodenal ileus	8	2	1	11
With mucous colitis ..	7	5	5	17
Total	169	44	26	239

In the small group of patients for whom right colopexy has been advised, the operation performed by one of us (A.E.L.) has consisted in appendicectomy followed by a modified Waugh's colopexy. An extraperitoneal cave is constructed in the right lateral abdominal wall, into which the caecum is dropped and carefully maintained in position by means of interrupted silk sutures placed both anteriorly and posteriorly. A number of opportunities have occurred to demonstrate the permanent fixation induced by this method.

The results of colopexy depend on the pre-operative success in determining that the syndrome under review is caused by a maximum of physical drag and a minimum of neurotic fixation. The majority of our patients claim that the operation was a brilliant success. Only one patient is sure he obtained no benefit, while in several others there is evidence that a transference of neurotic symptoms to other areas has been effected.

We are quite sure that this operation has a place in surgery and that in the future, as in the past, there will be a minority of patients, to whom, after simpler measures have failed, this operation should be offered in the certainty that by this means alone can their symptoms be relieved.

Summary.

1. Certain painful and dyspeptic syndromes possessing a postural pattern have been met with frequently in practice. Unsuccessful abdominal operations had already been performed on many of these patients.

2. The gradual recognition of the relation of the unfixed right colon to these syndromes is described.

3. The mechanism of pain production in the abdomen and abdominal wall is discussed, and reasons are given for regarding the lateral pain in right-sided visceroptosis as due to abdominal wall neuralgias.

4. The production of symptoms in right-sided visceroptosis is shown as due to (*a*) congenital non-fixation of the ascending colon, (*b*) a failure of secondary postural supports, and (*c*) a nervous facilitation.

5. Alternative explanations of the lateral pain in right-sided visceroptosis are discussed, and reasons are given for the non-acceptance of (*a*) chronic appendicitis, (*b*) orthopædic disorders of the back, (*c*) colonic dysfunction, (*d*) urological disorders, and (*e*) abdominal neurosis.

6. Treatment is directed towards the symptom-producing factors, especially the nervous facilitation and the postural failure. Colopexy is seldom advocated, but when effectively carried out for proper indications produces highly successful results.

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Reports of Cases.

AN UNUSUAL CAUSE OF DEATH.

By T. E. HOLCOMBE, M.B., B.S.,
Assistant Surgeon, Hornsby District Hospital.

A FEW days ago I was informed by telephone that a patient had been admitted to the Hornsby District Hospital in a serious condition; his temperature was subnormal; his pulse rate and respiratory rate were greatly accelerated, and he was obviously suffering from an acute condition of the abdomen. The cause of this was stated to be perforation of the bowel, following an enema.

On proceeding to the hospital I examined the patient. He was conscious and gave his age as forty-six years. He looked extremely ill and his body surface was cold and clammy. His pulse was feeble and the rate was rapid, and his respiratory rate was greatly accelerated. His abdomen was full and dull on percussion. There was a reddish mottling of the side on which he was lying.

When I questioned him he gave an unusual history. He had, it appeared, been suffering from hæmorrhoids for some years, and had for some considerable time been in the habit of replacing the presenting mass with an uncovered tennis ball. Some two or three days before he had replaced his piles so thoroughly that he had pushed the ball beyond reach—obviously beyond his sigmoid curve. He had informed no one, and had given himself enemata in an attempt to return the ball. Becoming ill, he had gone to a doctor, who sent him immediately to Hornsby District Hospital.

The operating theatre was prepared at once and it was decided to give him nitrous oxide and oxygen anæsthesia in view of his condition. However, in less than half an hour (just before he was to go to the operating theatre) he vomited copiously, a foul, fæcal-looking substance, and died.

A post-mortem examination some three hours later revealed an abdomen full of fluid and fæcal content. The descending colon was ruptured and an uncovered rubber ball was removed from the abdominal cavity, surrounded by omentum, suggesting that it had been lying free for some considerable time. His bowels had not opened since the accident, and apparently fluid was forced in but was blocked on its attempted return by the ball. Probably a combination of distension and pressure necrosis led to rupture.

This history is new to me, and I thought might be of interest.

Reviews.

CONGENITAL HEART DISEASE.

"CONGENITAL Heart Disease", by James Brown, is a clear exposition of a subject which was pioneered by Maude Abbott.[1] Her work in 1927, together with her subsequent investigations, which were so adequately illustrated at the centenary meeting of the British Medical Association in 1934, provided a firm framework from which the present author has built his structure after searching the diverse sources of information scattered through the medical literature and studying his subject with colleagues whose help he duly acknowledges.

In his introduction he calls attention to some of the special features of congenital heart disease. He then discusses embryology and morphology until he succeeds in illuminating the basic principles which will enable the student of the subject to understand the mechanism of the different types of congenital lesions.

He makes it clear that the classification of Abbott and Dawson is a most suitable one. Before considering those of no clinical significance he goes into details about those cases of clinical significance which come under the two headings: (*a*) non-cyanotic group, (*b*) cyanotic group.

The discussion of infective endocarditis, which has such a predilection for developmental flaws, rightly occupies some six pages. Special methods of investigation, including radiology, electrocardiography and angiocardiography, are then treated in a lucid manner.

A chapter dealing with differential diagnosis, general prognosis and treatment brings to its close a comprehensive review of a subject which, because of this book, will receive a greater amount of intelligent consideration in the future.

The extensive bibliography indicates the wide field of investigation covered by the author, who is to be congratulated not only on his grip of the subject, but also on the clarity of his exposition, which is greatly enhanced by numerous illustrative diagrams and clear photographs. The understanding of the various lesions is rendered easier by the foundation laid in the chapter on embryology and morphology.

By the time one has reached the end of this book the subject has assumed such an air of familiarity that one is well satisfied with the time spent in its study.

[1] "Congenital Heart Disease", by J. W. Brown, M.D., M.R.C.P.; 1939. London: John Bale Medical Publications Limited. Demy 8vo, pp. 270, with illustrations. Price: 21s. net.

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Extract from THE MEDICAL JOURNAL OF AUSTRALIA, *July 6, 1940.*

Analytical Department.

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THE DIAGNOSIS OF PULMONARY TUBERCULOSIS: AN IMPORTANT INVESTIGATION.

IN January of last year, under the title "Variations on an Old Theme", reference was made in these pages to the newer methods of diagnosis of pulmonary tuberculosis. While it was admitted on the one hand that the appearance of tubercle bacilli in the expectorated sputum in sufficient numbers to permit of their detection by the microscopic examination of films indicated that pulmonary tuberculosis had passed far beyond the incipient stage, it was also contended that the microscopic examination of smear preparations was at best a crude method for the disclosure of tubercle bacilli in sputum and should be supplemented by cultural methods of investigation, unless laboratory workers were content to allow the bacilli to elude them in approximately one-third of the specimens of sputum in which they were present. It was held that in capable hands the method of direct cultivation was a reliable and searching technique which should reveal the tubercle bacillus in little less than 100% of tuberculous exudates, with possibly the single exception of pleural effusion, and that the application of cultural methods and guinea-pig inoculation to gastric contents constituted a notable advance in the early diagnosis of pulmonary tuberculosis. In March of the same year the survey of the Second Australian Imperial Force by miniature radiography was described. It was pointed out that all men in whom evidence of pulmonary tuberculosis was discerned by miniature radiography were to be reexamined by the standard thoracic radiogram; they were then to be considered individually by carefully constituted medical boards, for whom the results of bacteriological examination would be made available.

The first report of this work has been made by Dr. Reginald Webster at a recent meeting of the Royal Australasian College of Physicians, held at Sydney. Dr. Webster gave an indication of the way in which the work was initiated, when he said that the systematic radiological examination of the entire personnel of the Second Australian Imperial Force, carried out under military discipline, created an opportunity unprecedented in this country to institute an important and far-reaching public health research. As a matter of fact, the whole event reflects the greatest credit on all concerned: on Dr. Webster, who conceived the idea of the research and had the will and the tenacity to carry it out; on Major-General R. M. Downes, who as Director-General of Medical Services saw at once the potentialities of the research and did everything in his power to further it; on Colonel C. Gordon Shaw, Deputy Director of Medical Services of the Southern Command, who gave his whole-hearted cooperation; and on the National Health and Medical Research Council, which voted a grant to make the work possible. According to the routine established in Victoria, recruits who displayed radiological evidence of pulmonary tuberculosis were paraded in batches of four, six or eight at the Children's Hospital, Melbourne. The men arrived in the morning, having had no food or drink since the previous evening. Those able to expectorate a specimen that could be regarded as genuine bronchial mucus, were not required to submit to the passage of the Rehfuss tube. Being young and apparently healthy men, many were unable to produce any sputum; from these the contents of the fasting stomach were aspirated, and the men cooperated well in the procedure. Dr. Webster thought that it was important that the men should present themselves in person and not send in samples of sputum; in this way unsatisfactory specimens were not received and risk of the substitution of specimens was avoided. The standards adopted were as follows: (*a*) If acid-fast bacilli were demonstrable in direct Ziehl-Neelsen preparations from sputum, a positive bacteriological finding was returned. (*b*) If an acid-fast bacillus, of the requisite cultural characteristics, was cultivated from the sputum of a man designated radiologically "active", it was accepted as the tubercle bacillus. (*c*) If an acid-fast bacillus was recovered from the sputum of a man, thought as a result of radiological examination to be the subject of old, healed, inactive or doubtful tuberculosis, the bacillus was required to produce progressive tuberculosis in a guinea-pig before final acceptance. In the meantime the report issued was one of a "strong presumption" of active tuberculosis. (*d*) Whenever the acid-fast bacillus was cultivated from the gastric mucus, irrespective of the nature of the radiological report, the virulence of the culture for the guinea-pig was determined. This was necessary because saprophytic acid-fast bacilli, some of which closely resemble true tubercle bacilli, are occasionally recovered from the stomach. The total number reported by Dr. Webster as having been examined was 322. Of these, 51 were sent in error for examination—they had been rejected or deferred by the radiologists, but on grounds other than those of suspected tuberculosis. This leaves a total of 271 men who were rejected or deferred because of X-ray evidence of pulmonary tuberculosis; these were subjected to bacteriological investigation. Of the 271, 100 or 36·9% were shown to be discharging tubercle bacilli. Of these 100 positive findings, 32 were made by direct smear examination of sputum (only one examination was made in each instance); 46 results were obtained by culture methods from sputum in which tubercle bacilli could not be detected by microscopic examination; 22 were

obtained by culture methods from the gastric mucus of men from whom no sputum was obtainable. The average time required for the reporting of sputum cultures was 18·6 days and for the reporting of gastric mucus cultures 21·6 days. Dr. Webster pointed out that the disparity between the figure, 32, representing the results obtained by examination of smears and that, 46, representing the number of cultures obtained could not be attributed to hurried or inadequate scrutiny—considerations of economy of effort and culture medium and the possibility of summary disposal of the specimen with an immediate positive report were strong inducements to persevere in the search for acid-fast bacilli in the films. Incidentally it should be mentioned that the method of cultivation applied was the sulphuric acid method of Lowenstein; the culture medium used was that of Petragnani as modified by S. Russell Jamieson. In every instance the positive findings following cultivation of gastric mucus were verified by guinea-pig injection, and in eight cases acid-fast bacilli, subsequently shown by cultivation and guinea-pig inoculation to be virulent tubercle bacilli, were detected by microscopic search of direct smears of the gastric mucus. Of the 100 men in regard to whom bacteriological proof of active tuberculosis was adduced, 54 maintained negative replies to all questions about hæmoptysis, pleurisy, sanatorium treatment and the occurrence of pulmonary tuberculosis among near relatives. An interesting sidelight on the mental attitude of intending recruits and on the need for the most careful questioning by medical examiners of recruits is afforded by the fact that of the remaining 46 men (who on enlistment had given a clear history) eight now admitted that they had suffered from hæmoptysis and sixteen had had an attack of genuine pleurisy; ten had formerly been in sanatoria and seventeen confessed that members of their own families or near relatives had suffered or were suffering from pulmonary tuberculosis. When nothing could be gained by further concealment they revealed the truth.

In regard to the correlation of radiological and bacteriological findings, Dr. Webster pointed out that in respect of a group of 152 men in whom the radiogram was looked on as indicating active pulmonary tuberculosis, the number of positive bacteriological findings was 83 or 54·6%. Further, among 90 men regarded after radiological examination as being the subjects of old, healed, inactive or doubtful tuberculosis, ten gave positive bacteriological results. Of these ten positive results, one was obtained by examination of a Ziehl-Neelsen preparation, six by culture from the sputum and three by culture from the gastric mucus. There is an important group of three men who gave no X-ray evidence of pulmonary tuberculosis, but who presented themselves fortuitously for bacteriological examination; in two of these instances diagnosis was made from a culture obtained from the sputum and in one by cultivation of the gastric mucus. This group is of great significance in the general appraisal of the subject.

The first point to be noted about this report is that it is a preliminary one, and the greatest satisfaction will be felt that the work is to be continued throughout the present year. At the same time it will have be realized that it will not be possible to make the work as complete as heretofore. Members of the Australian Imperial Force who had already been enlisted were paraded for this examination and there was no possibility of their avoiding it. In the future, when a diagnosis of active tuberculosis is made on radiological evidence and the candidate is to be refused admission to the army, it will merely be possible to suggest to him that he should complete the examination by visiting Dr. Webster; in this way some valuable information will no doubt be missed, but the modified routine rendered necessary by imperative Army requirements has already been operating sufficiently long to indicate that a steady flow of subjects for examination may be anticipated for the current year. That this work has already been, and will still be, of the greatest possible use to medical boards charged with the duty of accepting or rejecting army recruits is beyond question. In the wider sphere of general medicine it is clear that the medical profession will have to revise many of its ideas about so-called carriers of tuberculous infection; in other words, the current ideas of "open" and "closed" tuberculous infections will need to be revised, and in no half-hearted way. In the field of general medicine, too, physicians will have to realize what they have probably known but not always put into practice—that failure to discover tubercle bacilli in even several Ziehl-Neelsen preparations is not proof that the patient is not discharging the bacilli. Before the examiner can declare that he has done everything to determine this fact, he will need to undertake the searching investigations that Dr. Webster has described.

Current Comment.

WAR-TIME PRECAUTIONS FOR DIABETICS.

OWING to the frequency of air raids in Great Britain precautions of many different kinds have been taken, and among them are those that should be taken by diabetics. It is clear that persons who have to depend on large doses of insulin will face grave dangers if they are deprived of even one dose of the drug, and in conditions of aerial warfare this is quite likely to happen. Persons who need only small doses may do without their insulin for a day or two and suffer no harm, while those who have diabetes in a mild form and who do not need insulin will be in no danger if they have to do without food or are compelled to change their diet for a day or two. A special article on the subject of war-time precautions for diabetics, by R. D. Lawrence, was published in *The British Medical Journal* of September 7, 1940. Lawrence, as all readers of this journal know, is recognized throughout the English-speaking world as an authority on diabetes. The Director of the National Emergency Services in New South Wales has referred this article to the Medical Services Committee of that State, and it is as the result of a suggestion by this committee that the attention of readers is directed to the subject.

Lawrence's article, as are many of his well-known and effective writings, is presented as though it were intended for the diabetic patient; at the same time it is a clear and concise statement of what is necessary, and for this reason will be appreciated by medical practitioners. It is senseless for us to wait until a national emergency arises within the boundaries of this Commonwealth before we advise diabetics what personal preparations they should make in such an event. Presumably all diabetics pay periodical visits to a medical attendant, and it is suggested either that they should be advised of the kind of precautions that would be necessary for them or that they should be warned to seek instant advice when an emergency within these shores seemed inevitable.

In the first place, Australians may rest assured that there is little likelihood of a failure in insulin or of protamine zinc insulin supplies. The insulin made at the

Commonwealth Serum Laboratories alone is sufficient for Australian and New Zealand requirements. It is possible, as Lawrence suggests to his English readers, that local distribution would be temporarily upset; therefore, there is reason in his suggestion that patients taking insulin should have a month's supply in hand. He also states that diabetics should carry with them insulin and a syringe whenever there is the slightest possibility that they may not get home at the time appointed for the next injection; they should also carry a small supply of carbohydrate so that if the regular timing of meals is upset, enough carbohydrate can be eaten to balance the insulin and prevent hypoglycæmia. Dealing with the enforced omission of insulin or food, Lawrence points out that if a diabetic cannot get his usual insulin it is best for him as a general rule to continue to eat his usual carbohydrate, but no fat and only a little protein. By this means ketosis is more likely to be minimized than by complete starvation. A return to insulin and the usual diet will naturally be made as soon as possible. If a diabetic's supply of insulin is running short, it would, Lawrence thinks, probably be safe for a few days to economize by taking half the usual dose with the usual diet; but in these circumstances no fat should be eaten. If only a small amount of insulin is available, it is better to give it in three or four small doses than in one or two bigger doses. This, of course, does not apply to protamine insulin. Incidentally diabetics should not hesitate to use any brand of insulin if the one to which they are accustomed is not available. If insulin is available but no carbohydrate food can be obtained to balance it, the patient is in difficulties because the usual dose of insulin without food would cause hypoglycæmia and the complete omission of insulin would allow ketosis to develop. The subject of severe diabetes should take one-half of the usual dose of insulin without food. It is unlikely, Lawrence concludes, that food will be lacking for long, and as soon as it is available the usual meal and insulin must be taken, or only a half-dose of insulin if less than six hours have elapsed since the previous half-dose. It has also been suggested that in the interests of economy of bottles, labour and distribution, medical practitioners should prescribe 40-unit insulin instead of the 20-unit strength.

The main points of Lawrence's paper have been mentioned. Readers are advised to read the paper itself, for it is available in all parts of Australia. They will also find in *The British Medical Journal* of February 8, 1941, at page 203, a short account of a discussion on diabetics in war-time, held at a meeting of the Medical Society of London. At the gathering views similar to those put forward by Lawrence were expressed.

"ALCOHOLICS ANONYMOUS."

ABOUT six years ago an organization known as "Alcoholics Anonymous" was started in New York by chronic alcoholics who had a sincere desire to be cured of their addiction. Mention of this organization was made in the February, 1940, issue of the *Illinois Medical Journal* and in the December, 1940, issue of the same journal reference is made to it again. The organization is so unusual that readers of this journal will be interested to know something about it. The principles on which the organization is based are two in number. In the first place we are told that the chronic alcoholic can seldom be cured until he reaches a point at which he admits his inability to cope with his problem, and has in addition a sincere desire to achieve complete and lasting sobriety. In the second place the chronic alcoholic resents the efforts made by his relatives and friends to help him—he feels that they do not understand him or his problem; but when he takes a liking to persons who have themselves been drunkards he realizes that they understand because they have had the same personal experiences. Those who remember an anonymous article on chronic alcoholism published in this journal in December, 1934, by a general practitioner who achieved recovery after fourteen years of addiction, will remember the author's description of the "terrible depression, self-reproach and physical suffering" and the need for the "steadier". There is no doubt that the chronic alcoholic does not continue his vice because he likes it, any more than the morphine addict his injections for the same reason. Both are more to be pitied than blamed. Pity, however, does not help; when it passes into understanding help is possible. And when we come to think of it, who can understand so well as the man who has been through the hell that addiction brings and regained his self-control and ultimately his self-respect? Behind "Alcoholics Anonymous" there lies a great psychological truth—the help of self by the giving of help to others. The movement appears to work in "groups"; there are no officers—each member has equal standing—there are no dues, fees or expenses of any kind. A "new member" (presumably one who is seeking a cure) is introduced to the fellowship of the group. "'Business' gatherings are held weekly to talk over common problems. 'Social' gatherings are held several other nights of the week, where companionship is sincere and bridge, poker and conversation abound.". When a member has become thoroughly acquainted with the meaning of his new life he is supposed to go out himself and to work with other unfortunates. "The giving of himself, without thought of remuneration, gives him strength to combat his own desire." We must agree that "it is indeed a miracle" when a man who has been addicted to alcohol for years and in whom his friends have lost all confidence, will sit up all night with a "drunk" and at stated intervals administer a small amount of alcohol ordered by a doctor without taking a drop himself. But apparently this is what happens. The organization has 1,000 members, with groups in every large city in the United States. Of these people, who were hopeless alcoholics, 58% have attained complete sobriety and 92% "practical sobriety", whatever that means. A year ago Chicago had eight members; now there are 150. It will be admitted that the results are small; but many will agree that the idea behind this system should be fostered. And in any case it must be remembered that every alcoholic who recovers most likely brings happiness, or at least lack of suffering, to a whole family.

RETINITIS PIGMENTOSA.

THERE seems to be little doubt that *retinitis pigmentosa* is an hereditary abnormality; but authorities are still undecided as to the manner of its transmission. It is not known if the character is linked to one of the sex chromosomes, or whether it is dominant or recessive. One authority concluded that it could be either dominant or recessive, that the dominant was a special form of the degeneration, and that the recessive occurred in association with other hereditary abnormalities. It would seem that *retinitis pigmentosa*, being merely a degenerative process, can occur in a variety of forms and in company with a variety of other inherited abnormal characteristics. It is because of this that the confusion as to its compliance with the Mendelian laws has not yet been dissolved. It is possible that *retinitis pigmentosa* might sometimes occur in association with an inherited dominant characteristic and sometimes in association with a recessive. The wider recognition of the Laurence-Moon-Biedl syndrome (*retinitis pigmentosa*, polydactyly, hypogonadism, obesity and mental deficiency) has called more attention to the problem. The Laurence-Moon-Biedl syndrome is recognized as a Mendelian recessive. It is of interest to read a report by E. Cunningham Dax, who has been responsible for the study of a series of patients from among a large group of mental defectives.[1] In his opening remarks he points out that Zondek has demonstrated a disturbance of the metabolism of water and salt in *retinitis pigmentosa*, that Nagayama has discovered a thyreotrophic substance in the urine and that he (Dax) has found that the blood and urine of a person suffering from *retinitis pigmentosa* have a

[1] *The Quarterly Journal of Medicine*, October, 1940.

"melanosome-dispersing action on the skin of the frog". He draws attention to the coexistence with *retinitis pigmentosa* of abnormalities that might be attributed to disturbance of the diencephalon; but he points out that: "In considering some of the large number of associated defects which have been described, it is important to bear in mind that in more than two-thirds of the cases of retinitis pigmentosa collected by Bell no associated abnormality was noted either in the affected persons or their relatives."

The tendency to associate *retinitis pigmentosa* with disturbance of the hypophysis or the diencephalon probably arose only after the Laurence-Moon-Biedl syndrome had become well known; for it is generally recognized that the hypogenitalism and obesity of this syndrome are hypophyseal in origin. Zondek "believes that abnormal hormonal influences from the pituitary body are probably produced by some interference in its co-ordination with the hypothalamus, resulting in the eye condition, and that the pathological findings originate from a systemic destruction based on an inherited predisposition". Zondek and Koehler noted characteristics of diencephalic disturbance in persons suffering from pigmentary degeneration of the retina. The same authors suggested that the lesions of the *fundus oculi* in juvenile amaurotic idiocy were an atypical form of this degeneration. Dax quotes a number of other authorities to similar effect.

Among 1,200 mentally deficient patients Dax discovered only eleven who were afflicted with *retinitis pigmentosa*. He was able to obtain the family histories of ten of them, tracing 385 relatives. The first patient, affected with the Laurence-Moon-Biedl syndrome, was a woman whose stepsister and whose mother's maternal cousin (sex unknown) were insane. The mother, by the way, was one of a family of sixteen (two boys and fourteen girls). The second and third patients were also females who exhibited the characteristics of the Laurence-Moon-Biedl syndrome. Nothing was discoverable about the family history in Case II; but in Case III one of the patient's sisters suffered from failing vision. The fourth patient was also a woman; she was afflicted with obesity and microphthalmos. Her sister was mentally deficient; there was a history of insanity and alcoholism on her mother's side of the family. The fifth patient was a woman suffering from cerebellar hypoplasia, mental deficiency and hypogenitalism. One of her sisters suffered from mental deficiency and *retinitis pigmentosa*; her father was colour-blind and an alcoholic. There was no history of hereditary defect on the mother's side of the family. In Case VI the patient was a woman suffering from partial deaf-mutism. On the father's side there was a strong history of failing sight, defective speech, deafness and mental deficiency. Transmission was through the males. The seventh patient was also a female. She was afflicted with deaf-mutism, skeletal abnormalities and microphthalmos. The father was an alcoholic; the mother and maternal grandfather were insane, and the paternal grandmother was obese. The eighth patient, a male, was also afflicted with deaf-mutism. One brother was insane and an alcoholic; there was no other history of inherited defect. In Case IX the patient, a male, suffered from deaf-mutism and arachnodactyly. There was a history of obesity, mental deficiency and epilepsy on his mother's side, and hare-lip on his father's side. Two siblings suffered from *retinitis pigmentosa* without other defect; one was insane; one was mentally deficient; three had hare-lip, and one had long digits. One paternal cousin had hare-lip and one was insane. The parents were not related before marriage. The tenth patient, a male, suffered from schizophrenia and hypogenitalism. The father was syphilitic; a paternal uncle was mentally defective; a maternal uncle was an epileptic; a brother was insane. The eleventh patient was a male who suffered from *pes cavus*. No history of the paternal side of the family was obtained; no history of inherited defects was noted on the maternal side. The appearances of the *fundus oculi* were typical of *retinitis pigmentosa* in every case except Case VI, in which the pigment was "practically confined to linear deposits along the vessels". There were, of course, numerous other ocular abnormalities. It is notable that every patient but one, who had heterochromia, had grey or blue irides. Only three of the eleven patients were well proportioned. In the others, such abnormalities as comparative shortness of limbs, kyphosis and scapular winging, were noted. Every patient except the three mentioned as being well proportioned had some abnormality of the digits, in "length, breadth, relative size and position, shape, fusion, and number". In three cases the pituitary fossa was enlarged; in one it was very small.

The results of Dax's application of the sodium chloride excretion test and the urine dilution test did not agree with those obtained by Zondek. He found no gross variation from the normal. The majority of the patients suffered from a deficiency of vitamin *C*. The *pars intermedia* of the pituitary gland secretes a hormone that, when injected into the dorsal lymph sac of the frog, causes a pronounced darkening of the animal's skin. It is not found in any animal tissue excepting the pituitary; but at the beginning of menstruation, in pituitary disease, in pregnancy, in hyperthyreoidism, in migraine, and in some cases of cutaneous pigmentation it is found in the urine. Dax has found the same or a similar substance in the urine of persons affected with *retinitis pigmentosa*. He regards this as evidence of a diencephalic disturbance. Dax points out that a thermal centre has been shown to exist in the hypothalamus. This is of interest in the present argument, because three of his patients were found to have a mild degree of persistent hyperthermia. As further evidence of a central origin for *retinitis pigmentosa* he mentions that in three of his cases there was a diminished glucose tolerance.

This is an interesting and valuable report, the result of painstaking work and careful observation. It should be of value in the elucidation of the various problems associated with *retinitis pigmentosa* as well as other hereditary abnormalities. At the same time it should be borne in mind that all Dax's patients were mentally defective at the commencement of his study and they represent only a small percentage of a large group of mentally defective people. It might be argued that, just as an otherwise normal person might suffer from *retinitis pigmentosa*, so might a mentally defective person. It should be further noted that in discussing the Laurence-Moon-Biedl syndrome, no less an authority than Gorsby states: "The significance of the affection in the elucidation of the pathology of retinitis pigmentosa has been overrated, for the fundus lesion is not generally a typical primary degeneration of the retina. 'Atypical' retinitis pigmentosa is the rule, and occasionally the lesion is essentially a macular dystrophy with optic atrophy."

VITAMIN C AND THE HEALING OF WOUNDS.

IN our issue of November 9, 1940, reference was made in these columns to recent work on vitamin *C*. Among the contributions discussed was one by J. H. Crandon and C. C. Lund, who with D. B. Dill had undertaken a detailed investigation of the results of vitamin *C* deficiency in man. These workers observed *inter alia* that good normal healing of a wound might occur when the ascorbic acid value in the plasma had been zero for forty-eight days. On the other hand, failure of wound healing was very obvious when the ascorbic stores of the body generally were depleted, and microscopic evidence showed that lack of intercellular substance was largely responsible. Another paper by Lund and Crandon has recently been published together with one by other workers on plasma vitamin *C* and serum levels in wound disruption.[1] In their present paper Lund and Crandon cover much of the same ground as in the last; but it will be well to emphasize the observation that plasma determinations of the vitamin *C* level, if the findings are low, are not sufficient evidence on which to base treatment, as the great bulk of such

[1] *The Journal of the American Medical Association*, February 22, 1941.

patients are in no known danger from the deficiency. At the same time they insist, and most clinicians will agree, that a history of months or of years of inadequate intake is ample evidence on which to base treatment. They point out that there is evidence that most patients with scurvy are also suffering from multiple deficiencies, and they believe that if treatment is indicated at all it is probably wise to give large doses of other vitamins when large doses of vitamin *C* are given. One of their general conclusions is that vitamin *C* deficiency may be a factor in the failure of some human wounds to heal—their moderation should be noted. They state that a dietary history should be taken of all patients who come to major surgery, and if long-continued and pronounced deficiency of intake of vitamin *C* is found, ascorbic acid should be given.

The paper on plasma vitamin *C* and serum protein levels in wound disruption is by J. B. Hartzell, J. M. Winfield and J. L. Irvin. Their observations are full of interest. They refer to 1,458 cases in which wound disruption has occurred. Of these, 320, or 22%, took place in patients who had carcinoma, and 195, or 13·4%, in patients with gastric or duodenal ulcer. The nutritional condition of such patients is likely to be poor because of restricted diet, and the serum protein concentration to be low because of decreased production of proteolytic enzymes. Hartzell and his co-workers, in the course of some two hundred determinations, have found that persons suffering from carcinoma of the gastro-intestinal tract or from peptic ulcer almost always have low vitamin *C* and serum protein values. They have confirmed the observation made by other authors that some patients will manifest a fall in the vitamin *C* level after certain operative procedures; they also recall that the body in the presence of infection requires an increased intake of vitamin *C* to maintain a normal degree of saturation. They make the perfectly true statement that, generally speaking, if a blood transfusion is given, it is because the hæmoglobin value and the red cell count are low, rather than because of a low serum protein value; the vitamins are usually forgotten. To them it appears logical that if normal healing is to be expected, the ascorbic acid level and the serum protein level should be brought up to and kept normal.

The role of vitamin *C* in the healing of wounds has also been investigated by A. H. Hunt at the surgical unit of Saint Bartholomew's Hospital, London, whose paper is carefully documented and well illustrated with photomicrographs.[1] (Incidentally the reader cannot fail to remark on the first-class way in which this paper and others in the same journal are produced; it is a glowing tribute to the tenacity and the imperturbability of the sorely tried people of Great Britain.) Hunt points out that when clean sutured wounds break down in mysterious fashion in spite of meticulous care at the time of operation, there is usually evidence of a nutritional deficiency affecting the proliferating powers of the cells or the capacity of the mesodermal cells to lay down intercellular substances. In soft tissue scars fibroblasts proliferate and migrate among the fibrinous strands which seal the incisional gap during the first few days of wound healing, and the strength of a scar is dependent on the intercellular substance laid down by the fibroblasts and on the full maturation to collagen of the intercellular substance which is known as precollagen. Hunt mentions many authors who have shown that vitamin *C* has an effect on the healing of wounds in guinea-pigs. It is supposed, he states, that ascorbic acid acts as an oxidation reduction enzyme, enabling the deposition of reticulum, collagen, bone, enamel, dentine, and possibly cartilage and elastin to take place. "It has not yet been established whether the defect in vitamin *C* deficiency is due to a failure of the process of gellation of the intercellular matrix or to a degeneration of the specialized mesodermal cells of the supporting tissues." Hunt gives details of experimental investigations on guinea-pigs which show that a deficiency of ascorbic acid interferes with the healing of wounds. He reports an experiment on a young man, carried out after he had been on a scurvy-producing diet for three months; this experiment yielded no positive findings. He then reports observations made on human necropsy material taken from "an unselected series of 28 relevant cases". This sounds something like a contradiction, for if irrelevant cases were rejected the series could not really be called unselected. Be that as it may, the series is interesting and the disease for which operation was performed was in every instance one affecting the gastro-intestinal tract. The findings, which are set out in tabular form, need not be described. It must suffice to state that of 18 cases in which death occurred after the seventh day, eight had precollagen in place of collagen. In all these cases there was good indication that there had been in the past an insufficient intake of vitamin *C*. This small group of eight cases included all the five cases in which abdominal disruption had occurred in the absence of gross infection; in three cases also leakage had occurred at lines of suture without the occurrence of local necrosis. Hunt thinks that, taken in conjunction with his animal experiments, the observations on human wounds are suggestive that a partial deficiency of vitamin *C* produces effects which are as important in the healing of human wounds as in those of guinea-pigs. Peculiarly he attacks only one aspect of the failure in wound healing. He thinks it important, however, to mention factors other than vitamin *C* deficiency, and of these reference must be made to hypoproteinæmia. Hartzell and his collaborators pay special attention to it. Hunt states that hypoproteinæmia is partly responsible for lack of cellular response in the healing of wounds, and he makes much the same point as Hartzell and his co-workers make, for he states that when fluids are given by the rectum or intravenously there is a tendency to give blood only when the hæmoglobin falls below a certain arbitrary level. In these circumstances hypoproteinæmia sometimes starts after intravenous therapy has been begun and in the absence of anæmia. For this reason the blood protein should be repeatedly estimated and maintained. Further than this he does not go in the question of hypoproteinæmia.

In his general discussion Hunt rightly insists that it is of no scientific value to state that wound disruption has been reduced by about 75% since the routine administration of ascorbic acid has been adopted for all major operations. This, however, is what he has found; moreover, leakage from suture lines has occurred once only in a large number of operations during the past thirty months. Though he is careful to state that vitamin *C* is not a panacea in the healing of wounds, he recommends that it should be administered: (*a*) when clean and quick-healing of the wound is particularly desirable, (*b*) in major abdominal operations, (*c*) when a hollow viscus has been opened, (*d*) when post-operative complications are anticipated, (*e*) when there is evidence of a nutritional deficiency, (*f*) in all cases of serious injury. In cases such as these he thinks that saturation should be produced if possible before operation. In emergencies saturation should be reached before the seventh day. Saturation can be produced even in a grossly deficient patient by doses of a thousand milligrammes of ascorbic acid given every day for three days, and a daily dose of one hundred milligrammes will maintain it.

A general criticism may be levelled at Hunt's recommendations that they are too sweeping, especially in view of the work of Lund and Crandon and of the fact that he has not on his own showing paid much attention to the serum protein value. It may be that the community in which Hunt's surgery was carried out was in the habit of taking food particularly deficient in vitamin *C*. Such wide indications for saturation with the vitamin would, we believe, not be necessary in Australia. This does not, of course, mean that the subject can or should be lightly dismissed by Australian surgeons. It would in fact be worth while if investigations in the subject could be carried out in this country. One thing is quite certain, and with this every surgeon worthy of the name will agree, inquiries into vitamin *C* concentration and protein level in the blood must not be allowed to divert attention from meticulous care in surgical technique. Biochemical adventures in everyday practice will never serve as a cloak to surgical incompetence.

[1] *The British Journal of Surgery*, January, 1941

Abstracts from Medical Literature.

PATHOLOGY.

Ætiology of Acute Hæmorrhagic Pancreatitis.

C. J. SMYTH (*Archives of Pathology*, September, 1940) examines the current theories of the genesis of acute hæmorrhagic pancreatitis. He comes to the conclusion that none of them adequately explains the production or the mechanism of the disease, although there is abundant evidence in the literature to show that vascular disturbances play an important ætiological role in many instances. It is evident to the author that the solution to the problem of the genesis of this disease will be found in experimental studies in which the earliest stages of the disease can be investigated, rather than in review of material obtained at autopsy. Localized areas of acute pancreatic necrosis were uniformly produced in 21 dogs by the injection of mercury into the pancreatic artery; but none of these animals died of a spreading type of acute pancreatic necrosis. The frequent production of the "specific" vascular lesion described by Rich and Duff in 1936 indicates that this lesion is a result of, or concomitant with, the disease; it is not of ætiological significance. In these dogs small areas of pancreatic necrosis remained localized, even though the adjacent normal acini were stimuated to maximum secretory function by food or by acetylbetamethylcholine chloride and physostigmine.

Relation of Anatomical Pattern to Pathological Conditions of the Coronary Arteries.

MONROE J. SCHLESINGER (*Archives of Pathology*, July, 1940) has studied the coronary artery pattern in 225 hearts from patients over thirty-five years of age. He has found that it is not the same in all its details in any two human hearts. These multitudinous variations can be classified into three definitely different, distinctive anatomical and functional groups. In group 1, comprising 48% of human hearts, the right coronary artery predominates in the blood supply of the heart; these hearts are intermediate to the other two groups in their reaction to the ravages of coronary arteriosclerosis. In group 2, comprising 34% of human hearts, the coronary artery blood supply of the heart is balanced between the right and left coronary arteries; these hearts suffer the least from the effects of coronary arteriosclerosis. In group 3, comprising 18% of human hearts, the left coronary artery predominates in the blood supply of the heart; these hearts suffer the most from the effects of coronary arteriosclerosis.

Pathology of Acute and of Healed Experimental Pyelonephritis.

PYELONEPHRITIS has been recognized for years as an acute inflammatory disease involving the renal parenchyma and pelvis. It has become increasingly important to the physician, because it must be considered not only as a disease manifesting itself by signs of an inflammatory process, but also as one which in its chronic and healed stages is an important cause of hypertension and renal impairment. Pyelonephritis, chronic or healed, is a common cause of Bright's disease, the cases forming a larger proportion of that group than cases of glomerulonephritis. It is therefore of considerable importance to be able to recognize accurately the end results of a pyelonephritic process in the kidney. In man it is difficult to trace step by step the development of such a process because of the difficulty of obtaining very early and healing lesions. Consequently, G. K. Mallory, A. R. Crane and J. E. Edwards (*Archives of Pathology*, July, 1940) have thought it worth while to produce acute pyelonephritis in animals and to trace the renal lesions from the earliest recognizable process to the completely healed stage. This was done by injecting colon bacilli intravenously into rabbits which had partial ligation of one ureter. Acute pyelonephritis similar to that seen in man was produced in the obstructed kidney in about 75% of the animals. Extensive pyelonephritis was never found in the unobstructed kidney. By release of the ureteral obstruction at the end of four to five days after the injection of bacilli, healing of the pyelonephritic process was induced. In this way stages of acute, healing and healed pyelonephritis were produced. The acute lesions were found to arise (*a*) from interstitial abscesses starting around clumps of bacteria in the small blood vessels, and (*b*) as a result of organisms passing through the glomerulus into the tubules. In the healed lesions thyreoid-like areas of colloid casts were found. In earlier lesions a fusion of disintegrating polymorphonuclear leucocytes was found to lead to the formation of these casts. The gross and the histological appearance of the healed lesions confirm the criteria used in the diagnosis of healed pyelonephritis in man, that is, groups of tubules the lumens of which were filled with colloid casts staining from blue to pink, cortical fibrosis with focal lymphocytic infiltration and disappearance of glomeruli and tubules, and similar fibrosis and infiltration in the pyramids and around the pelvis.

Genesis of Hydatidiform Mole.

ARTHUR T. HERTIG AND HENRY W. EDMONDS (*Archives of Pathology*, July, 1940) have studied a series of 1,027 spontaneously aborted ova and 74 hydatidiform moles with respect to the genesis of hydatidiform degeneration. Hydatidiform degeneration to some degree is common, having occurred in 40% of these spontaneously aborted ova, or in 4% of all pregnancies. Typical stages in the evolution of a hydatidiform mole are described. A classical hydatidiform mole is uncommon, occurring only once in 2,062 full-term deliveries in the authors' clinic. Pathological ova, of ten and two-tenths weeks' mean menstrual age, constituted 47·4% of the total number of spontaneously aborted ova; 66·9% of the pathological ova showed early hydatidiform degeneration of the chorionic villi. "Nonpathologic" ova, of fifteen and four-tenths weeks' mean menstrual age, constituted 52·6% of the total; 11·6% of such ova showed early hydatidiform degeneration of their villi. Hydatidiform degeneration of the chorionic villi of pathological ova begins in all probability at about the fifth week of pregnancy, the time when the fœtal circulation should begin to function. The fœtal circulation in the chorion of the pathological ovum fails to function because of extreme defectiveness or absence of the embryo. The vascular *Anlagen* disappear coincidently with the onset of hydatidiform degeneration, both processes being a function of absence or of defectiveness of the circulation. Hydatidiform degeneration is prone to occur in the villi of early pathological ova because the stroma of the villi is normally loose and their chorionic epithelium is normally active. Hydatidiform degeneration in the villi of early pathological ova is an expression of continued physiological activity of the trophoblast (absorption and/or secretion) with resultant accumulation of intravillous fluid, which cannot be utilized because of the lack of a functioning fœtal circulation. Hydatidiform degeneration is less apt to occur in "nonpathologic" ova, because of a functioning fœtal circulation of some weeks' duration, relatively dense stroma and relatively inactive chorionic epithelium. Transitional moles, of sixteen and six-tenths weeks' mean menstrual age, are usually typical pathological ova containing no embryos or at most extremely defective ones, although a few contain macerated normal embryos. Typical hydatidiform moles, of seventeen and four-tenths weeks' mean menstrual age, are true pathological ova possessing no embryos or rarely a very defective embryo. A typical hydatidiform mole is therefore derived from a true pathological ovum in which the embryo was either absent or very defective from the beginning and which, for reasons unknown, failed to abort at the usual time. Hence it constitutes a type of "missed abortion".

Generalized Sarcoidosis of Boeck Accompanied by Tuberculosis and Streptococcal Bacteriæmia.

WILLIAM F. HOLLISTER AND GEORGE T. HARRELL (*Archives of Pathology*, February, 1941) report a case of generalized Boeck's sarcoid that occurred in a patient who had been studied clinically for two years. He died suddenly and an autopsy was performed. The lesions of sarcoid were found in nearly all the organs, including a joint capsule and the epiglottis, where they rarely are reported. Several caseous lesions were found in the liver and spleen. The twenty-two recorded autopsies are reviewed briefly. Tubercle bacilli were recovered in guinea-pigs inoculated with material from a lymph node from the hilus of the liver. None were recovered from animals inoculated with femoral node, tonsil, lung, spinal fluid and blood. The authors do not think that their observation has solved the question whether the sarcoid is of tuberculous origin or not. According to them the patient may have had two diseases: localized tuberculosis, from which organisms were recovered, and sarcoid, the ætiological agent of which remains unidentified. A simultaneous streptococcal bacteriæmia was thought not to be significant.

Fœtal Endocarditis.

PAUL GROSS (*Archives of Pathology*, February, 1941) reports on stenosis of the mitral and aortic valves which occurred in a full-term new-born boy and which resembled strikingly the rheumatic valvular disease of adult life. This condition was associated with hypoplasia of the root of the aorta, rudimentary development of the left ventricular cavity, the presence of a large patent *ductus arteriosus* and diffuse parietal endocardial sclerosis of

the left ventricle. The genesis of these lesions is considered non-inflammatory because of the lack of inflammatory residua. In general, the author is of the opinion that the occurrence of fœtal endocarditis has never been established. The macroscopic and microscopic abnormalities considered criteria of fœtal endocarditis have been observed also in the presence of congenital cardiac defects which, after study, have been diagnosed as non-inflammatory lesions. The assumed ætiological factor in the reported cases of fœtal endocarditis has been either anamnestically absent or variable in identity. The myocardial lesions usually seen in so-called fœtal endocarditis closely resemble healed bland infarcts. Such infarctions may be related to the obliteration of arterio-luminal, arterio-sinusoidal and Thebesian vessels by the parietal endocardial fibrosis. The valvular and parietal endocardial changes seen in these cases are better explained on the basis of a developmental defect, since they show no inflammatory residua.

The Giant Cells in Riedel's Struma.

ACCORDING to E. Goetsch (*Archives of Surgery*, August, 1940), the occurrence of giant cells in cases of chronic non-specific thyreoiditis, usually designated as Riedel's struma, has been noted in a considerable number of reports, but few studies have been made with the particular purpose of determining their mode of origin and evolution, their possible function, if any, and their ultimate fate. As a result, they are little understood and various substantiated views have been expressed regarding their origin and significance. They are, in the author's opinion, most commonly misinterpreted as foreign body giant cells possessing phagocytic properties. He has, therefore, very thoroughly examined a case of Riedel's struma and comes to the conclusion that the so-called giant cells are formed by fusion of follicular epithelial cells into multinucleated, syncytial masses of protoplasm which are desquamated into the lumen of the follicle. These giant cells represent secretory thyreoid cells which are on the way to destruction, as is indicated by the readily recognizable transitional stages in their progressive degeneration, final disintegration and eventual disappearance. His observations indicate that the protoplasmic giant cell masses seen in Riedel's struma do not possess phagocytic properties, as has heretofore been generally assumed, and should therefore not be considered true foreign body giant cells. Ultimate understanding of the ætiological factors causing the destruction of the thyreoid parenchyma in cases of Riedel's struma will, according to him, be advanced by realization that the so-called giant cells represent merely a first stage in the disintegration and disappearance of the thyreoid epithelium. They are not causes, but rather effects of the as yet obscure destructive influences operating in this disease.

A Hæmocytometer Method for Reticulocyte and Platelet Counts.

W. J. SALTER (*The Journal of Pathology and Bacteriology*, January, 1941) describes a hæmocytometer technique he has used for the past five years. It originated as a modification of a method described by Cooke for platelet counts, but was found to be equally valuable as a method for counting reticulocytes. Both these elements can be conveniently counted in a hæmocytometer chamber, and the results obtained suggest that the method is both accurate and rapid. The diluting fluid is prepared by the mixing of two parts of an aqueous solution of brilliant cresyl blue (1 in 300) with three parts of a 1 in 1,400 aqueous solution of potassium cyanide, the final solution being centrifuged at high speed for five minutes, because the smallest amount of stain deposit in the fluid makes it difficult to count either type of cell. The two solutions must be kept separately and mixed immediately before use. Blood is taken up to the mark 0·5 in a white-cell pipette, diluted to mark 11 with the diluting fluid and well shaken. A drop is then placed in the counting chamber and, after five to ten minutes, the count is made. Platelets appear as ovoid blue or lilac-coloured bodies. Reticulocytes are recognized by the characteristic reticular structure inside shadowy red cells. A leucocyte count can be made at the same time. The number of platelets may be calculated directly from the hæmocytometer count, but in order to ascertain the percentage of reticulocytes present in the blood a red cell count must also be carried out.

MORPHOLOGY.

The Mechanics of the Carpus.

M. A. MACCONAILL (*The Journal of Anatomy*, January, 1941) has tried to correlate the structure of the carpus with observations on its functions. He considers that side-to-side pressures between the carpal bones are as important functionally as longitudinal pressures, and believes that removal of the lunate destroys this lateral pressure, thus weakening the wrist. The author regards the proximal row of carpals as composing two elements—the lunate is one, the remaining carpals the other. In full dorsiflexion the two elements are virtually united; in other positions they fall apart. Thus he concludes that the remaining carpals act like a vice to lock the lunate in dorsiflexion, and he quotes observations on the weakness of dorsiflexion after removal of the lunate.

Digital Ligaments.

J. GRAYSON (*The Journal of Anatomy*, January, 1941) has investigated the cutaneous ligaments of the digits, first described by Cleland. These ligaments extend laterally from each side of the phalanges to be attached to the skin, thus forming a sort of septum dividing the side of the finger into anterior and posterior portions. The digital vessels and nerves lie anterior to the septum, which appears to hold the skin taut in flexion of the fingers. In addition, the author was able to show that there is another septum lying anterior to the vessels and nerves. Therefore, he refers to Cleland's ligaments as the deep digital skin retinacula and to his own as the superficial or volar retinacula. The volar retinacula arise from the tendon sheath, and there is a pair—proximal and distal—to the proximal interphalangeal joint and only one to the distal interphalangeal joint. The author considers the possibility that these retinacula are derived from the same fibrous layer as produces the primitive interdigital web, and he found some confirmation of this in the manus of *Ornithorhynchus*.

The Thalamus in the Phalanger.

F. GOLDBY (*The Journal of Anatomy*, January, 1941) offers a detailed account of the thalamus in the phalanger as the basis for future experimental work. He finds that on the whole the thalamus in this animal conforms closely to the pattern found in other primitive mammals; but in some respects there are distinct advances. These are revealed in the fact that the cells of the dorsal nucleus of the lateral geniculate body betray a definite layering; the *nucleus lateralis posterior* (pulvinar) is differentiated into two nuclei; there is differentiation of the lateral part of the dorso-medial nucleus; and there is a possible representative of an arcuate nucleus in the ventral group. The author considers these changes similar to those found in the shrew, *Tupaia*.

Cutaneous Tension Lines and Hair Tracts.

F. WOOD JONES (*The Journal of Anatomy*, January, 1941) considers the possible correlation between the disposition lines in the skin and the distribution of the hair tracts. The tension lines are due to the cleavage lines (*Spaltbarkeitslinien*) produced by the fibrous tissue strands in the skin. The view has been advanced that the arrangement of the hair tracts is imposed by the disposition of the cleavage lines; but the author shows that on the whole there is no evidence of the conformity between cleavage lines and hair tracts that the view would demand.

Ascorbic Acid in the Early Embryo.

S. A. BARNETT AND G. BOURNE (*The Journal of Anatomy*, January, 1941) have used the acid silver nitrate method to determine the distribution of vitamin *C* in chick embryos up to the fourth day of incubation. Ascorbic acid was found in association with intracellular and extracellular yolk granules in the blastoderm before incubation, and after twelve and eighteen hours' incubation. In embryos from the first to the third day ascorbic acid was found only in extraembryonic tissues, especially ectoderm and endoderm. By the fourth day the acid appeared in the central nervous system, the liver and pancreatic diverticula, and in some mesodermal derivatives.

Accessory Tymus Tissue.

J. H. VAN DYKE (*The Anatomical Record*, February, 1941) discusses the possible development of thymic tissue from the fourth pharyngeal complex (fourth pouch, ventral diverticulum, parathyreoid IV and the ultimobranchial body). He reviews previous accounts of 23 cases of "thymus IV" and presents the results of his own study of 24 human fœtuses of late development. In 42% of these specimens there was a thymus IV; it is usually late in development and variable in size, but typical in structure and very vascular. The accessory thymus is usually adjacent to, or in continuity with, parathyreoid IV or the thyreoid gland, and the thymic epithelium is probably derived from the epithelium of the fourth pouch. Apparently the ultimo-branchial body disappears.

The Royal Australasian College of Physicians.

COUNCIL MEETING AND ANNUAL MEETING.

THE third annual meeting of the Royal Australasian College of Physicians was held in Sydney on Friday and Saturday, April 4 and 5, 1941. The meeting was well attended by New South Wales Fellows and Members. War conditions have inevitably reduced attendance by Fellows and Members from other States.

Council Meeting.

A meeting of the Council of the College was held on the morning of Friday, April 4, and on the afternoon of Saturday, April 5. Those present were: the President (Dr. S. V. Sewell), Sir Charles Blackburn, Sir Trent Champion de Crespigny, Dr. S. O. Cowen, Dr. O. A. Diethelm, Dr. A. W. Holmes à Court, Dr. F. S. Hone, Dr. Konrad Hiller, Dr. Alex. P. Murphy, Dr. F. J. Niall, Dr. H. J. Ritchie, Dr. M. D. Silberberg, Dr. S. A. Smith, Dr. A. H. Tebbutt, Dr. A. E. Rowden White and Dr. G. C. Willcocks (representing Dr. C. T. Hand Newton of New Zealand). Dr. A. J. Collins was appointed to the Council *vice* Dr. Allan Walker (on active service).

Admission of New Members.

At the meeting of the Council, the President admitted Dr. John Maude, of Sydney, and Dr. N. V. Youngman, of Brisbane, to membership of the College. Dr. Maude and Dr. Youngman were successful at the examination for membership which preceded the meeting. Dr. James F. Hughes (Victoria), Dr. R. L. Kenihan (South Australia), Dr. Garnet Leary (Victoria) and Dr. J. F. Meehan (Victoria) also passed the membership examination and will be admitted at the next meeting of the College in Melbourne.

Resignation.

The resignation of Dr. William Ray, of Adelaide, South Australia, was accepted by the Council.

Annual General Meeting.

The annual general meeting was held in the Stawell Hall of the College on Friday, April 4. The following Councillors were reelected: Dr. L. S. Latham (Victoria), Lieutenant-Colonel C. G. McDonald (New South Wales, on active service), Dr. Alex. P. Murphy (Queensland) and Dr. M. D. Silberberg (Victoria).

It was announced that so far as was known, fifty-eight Fellows and thirty Members of the College had proceeded or were about to proceed on service at the date of the meeting.

Scientific Programme.

The following contributions were given at the scientific sessions: "Bacteriological Examinations Supplementing the Radiological Survey of the Australian Imperial Forces", by Dr. Reginald Webster (Melbourne); "Hypoglycæmia", by Dr. F. S. Hansman (Sydney); "Some Problems of Preventive Medicine", by Professor H. K. Ward (Sydney); "Disuse Atrophy of Muscle", by Dr. J. C. Eccles (Sydney); "Infectious Encephalomyelitis", by Dr. S. A. Smith (Sydney); and "A Case of Acute Disseminated Sclerosis", by Dr. F. B. Byrom (Sydney). An interesting discussion followed each of the contributions.

Pathological exhibits were shown by Dr. A. D. Gillies (Sydney) and Dr. F. B. Byrom (Sydney).

On the evening of Friday, April 4, a joint scientific meeting of the Royal Australasian College of Physicians and the Royal Australasian College of Surgeons was held in the Stawell Hall of the Royal Australasian College of Physicians. Professor Eric Ashby, professor of botany at the University of Sydney, delivered a very interesting lecture entitled "Hormones in Plants". The lecturer brought forward much matter that was novel but of considerable interest to the medical profession, and in the discussion which followed he answered many questions.

A meeting of the joint Councils of the Royal Australasian College of Physicians and the Royal Australasian College of Surgeons was held on the afternoon of Saturday, April 5. Reports were received on the funds for assistance to consultants and specialists on active service from the members of the joint committees in several States and also on the subject of public relations of the Colleges.

Post-Graduate Work.

ANNUAL REFRESHER COURSE IN BRISBANE.

THE Queensland Post-Graduate Committee announces that a post-graduate course will be held at Brisbane from May 30 to June 6, 1941. The programme is as follows.

Friday, May 30, 1941.

4.30 to 5.30 p.m.—Technique of blood transfusion and intravenous medication, by the staff of the Brisbane Hospital, at the Brisbane General Hospital.

8.15 p.m.—Symposium on preventive medicine: Dr. S. F. McDonald, "Insect-Borne Diseases"; Dr. E. H. Derrick, "Prophylaxis by Inoculation"; Major N. M. Gutteridge, "Field Work and other Preventive Measures". At the Medical School, Herston Road, Brisbane.

Saturday, May 31, 1941.

9 a.m. to 11 a.m.—"Practical and Plaster Fracture Technique", by Dr. A. V. Meehan and Dr. J. R. S. Lahz. At the Mater Misericordiæ Hospital, South Brisbane.

11.30 a.m.—"Remedial Exercises", by Dr. Harold Crawford. At the Massage Department, Brisbane Hospital.

2.30 to 5 p.m.—Clinical meeting at the Mater Misericordiæ Hospital, South Brisbane.

8.15 p.m.—Surgical symposium: Dr. Alan E. Lee, "Delayed Closure of Wounds"; Dr. Neville G. Sutton, "Nerve and Tendon Suture"; Dr. G. H. Brandis, "Colostomy and its Closure"; Dr. H. J. Windsor, "Some Problems in Gastro-Duodenal Surgery". At the Medical School, Herston Road, Brisbane.

Sunday, June 1, 1941.

10 a.m. to 12.30 p.m.—Clinical meeting at the Brisbane General Hospital.

Monday, June 2, 1941.

9.30 a.m.—Demonstration on infant welfare at the Child Welfare Centre, Alfred Street, Valley. Each demonstration is limited to four members.

4.30 p.m.—"Prevention and Treatment of Venereal Diseases in the Forces", by Dr. V. N. B. Willis and Dr. G. S. S. Hayes. At the Medical School, Herston Road, Brisbane.

8.15 p.m.—Symposium on war neuroses: Professor J. Bostock, "Preliminary Remarks: The Nature and Causes of War Neuroses and Allied Conditions"; Major N. W. Markwell, "The Prevention of War Neuroses"; Lieutenant-Colonel B. F. R. Stafford, "The Treatment of War Neuroses". At the Medical School, Herston Road, Brisbane.

Tuesday, June 3, 1941.

9.30 a.m.—Demonstration on infant welfare at the Child Welfare Centre, Alfred Street, Valley. Each demonstration is limited to four members.

8.15 p.m.—"Fractures of the Wrist Joint", by Dr. G. A. C. Douglas, and "Compound Fractures of the Femur", by Dr. A. V. Meehan. At the Medical School, Herston Road, Brisbane.

Wednesday, June 4, 1941.

9.30 a.m.—Demonstration on infant welfare at the Child Welfare Centre, Alfred Street, Valley. Each demonstration is limited to four members.

11 a.m.—Pathological demonstration (renal disease), by Professor J. V. Duhig. At the Medical School Herston Road, Brisbane.

4 p.m.—Demonstration of medical cases by Dr. Hume Turnbull, at the Brisbane General Hospital.

8.15 p.m.—Symposium on tropical military hygiene: Professor D. H. K. Lee, "Selection for Tropical Service"; Sir Raphael Cilento, "The Malarial Problem in Malaya"; Colonel D. Gifford Croll, "The Military Significance of Tropical Diseases". At the Medical School, Herston Road, Brisbane.

Thursday, June 5, 1941.

8.15 p.m.—An address will be delivered by Dr. Hugh R. G. Poate to the Fellows of the Royal Australasian College of Surgeons at the Medical School, the University of Queensland, on the subject of "Diverticulosis and Diverticulitis". All members of the British Medical Association are cordially invited to attend this lecture.

Friday, June 6, 1941.

2.30 p.m.—Conference of the Council of the Queensland Branch of the British Medical Association with delegates from the Local Medical Associations at British Medical Association House, 225 Wickham Terrace, Brisbane.

8.15 p.m.—Joseph Bancroft Memorial Lecture: "Fear as a Factor in Disease", by Dr. Hume Turnbull, at the Medical School, Herston Road, Brisbane.

Members are informed that owing to the present military situation alterations may have to be made in the programme. The fee for the course will be one guinea.

NOTICE.

THE New South Wales Post-Graduate Committee in Medicine announces that the next library seminar arranged by the Post-Graduate Directors of Medicine, Surgery and Pathology will be held at the Prince Henry Hospital, Little Bay, New South Wales, on Monday, May 12, 1941, at 4.30 o'clock p.m. The subjects will be: (*a*) "Diaphragmatic Hernia", (*b*) "Parathyreoid Tumours". A cordial invitation to be present is extended to all medical practitioners.

Correspondence.

THE MEDICAL PROFESSION AND LEADERSHIP.

SIR: The article in THE MEDICAL JOURNAL OF AUSTRALIA on April 26 is very much to the point and demands the careful consideration of every member of the medical profession. We are living in a rapidly changing world and the future is to an extent unpredictable, as there is no previous experience to guide us. No danger like that existing at present has ever threatened the world before. It means, if the dictators succeed, the overturning of civilization. The nearest approach to it was in the Roman Empire in the fourth century A.D. The Roman citizen might well have been apprehensive, but thought our civilization has lasted for centuries and must continue. It collapsed and was followed by centuries of the dark ages. All that we can predict now is uncertainty and trouble.

If, as your article suggests, the medical profession rises to the occasion, they can, with their biological training and knowledge of human nature, play a most important part in guiding social activities, pointing out dangers with courage, and in general showing sympathy and knowledge in the confusion which must follow the war.

There is hardly a social activity which cannot be assisted and guided by the profession with their wide knowledge.

It is our chance to show that we are not selfishly considering only our own interests, but willing to assist the State in recovery and stabilization. The road will be hard and may take very many years to travel, and it is certain that new developments of many kinds will make their appearance.

The utterances of President Roosevelt and Mr. Cordell Hull show clearly how accurately they realize the altered international condition in trade and political relations which must be faced, and that they are convinced that stability will not be reached on narrow modes of thought.

It is our chance of rendering service and earning respect and confidence, as your article indicates. There is a Bantu proverb which is appropriate: "The leopard comes once to me but if you do not hold him he never comes again."

Yours, etc.,

JAMES W. BARRETT.

105, Collins Street,
Melbourne, C.1.
May 2, 1941.

SIR: May I congratulate you on the excellent editorial on "The Medical Profession and Leadership". The attitude of mind which expects every man to be a mere cobbler, sticking to his own last, is precisely that which has led to our present world chaos. We have accepted the material wonders of our civilization at their face value. We have allowed it to be so complicated that almost every intelligent man must stick to his own last to make a living.

Need we wonder that the lack of coordination of the units of society leads to chaos on the whole.

May I echo your sentiment in that we must be "up and doing". We must be not merely learning the elements of citizenship, but actively assisting in the inception of a new community order.

Yours, etc.,

JOHN BOSTOCK.

Brisbane Clinic,
Wickham Terrace,
Brisbane.
April 28, 1941.

COMBINED PREGNANCY.

SIR: Mrs. W., aged thirty years, two children, aged six, four years, was attending me for pregnancy. She was three months from her last period when she miscarried. I examined the fœtus, which was nine centimetres in length. She went to hospital and was curetted and subsequently went home. One month later, owing to her having repeated attacks of lower abdominal colic, she was operated on and a leaking ectopic pregnancy found and removed. This fœtus was twenty centimetres in length and a probable twin of the intrauterine one. The day after the operation she commenced to menstruate for the first time for over four months.

Parry states (Whitridge Williams's "Obstetrics", page 700) that 22 out of the 500 cases of tubal pregnancy collected by him were complicated by a coexisting intrauterine pregnancy. He designated the condition as "combined pregnancy".

Yours, etc.,

F. W. SIMPSON.

Wembley Park,
Western Australia,
April 5, 1941.

INTERESTING USES OF HYDRARGYRUM CUM CRETA.

SIR: Mrs. H. had four consecutive stillbirths, each at about nine months' gestation. The Wassermann test produced no reaction. Apparently in each case the fœtus died as a result of some change in the placenta. When the patient again became pregnant she took for about seven months, three times a day, tablets of *Hydrargyrum cum Creta*, one-quarter of a grain, and gave birth to a healthy infant at nine months' gestation. In the next six years she had three other infants, and had the same treatment and the same result each time.

Mrs. E., a *primipara*, aged twenty-eight years, had a normal gestation and gave birth to a full-time infant. The placenta was adherent and manual removal was required. During her next pregnancy she took for seven and a half months *Hydrargyrum cum Creta*, one-quarter of a grain three times a day. She had a normal gestation and the placenta separated normally.

Mrs. D., aged twenty-seven years, a *primipara*, had a normal gestation but a difficult labour. The placenta was strongly adherent and manual removal was necessary. During her next pregnancy she took for about eight months *Hydrargyrum cum Creta*, one-quarter of a grain three times a day. The gestation and labour were normal. The placenta separated normally. The history of this patient's mother is interesting for comparison: she had three infants, and all the placentæ were adherent and required manual removal, but she had no treatment.

I may add that in all successive pregnancies after one in which the placenta has been adherent I use this treatment as a routine measure, and I have never known it to fail. I have had many Wassermann tests performed, and none has produced a reaction.

These cases are reported in view of the increasing interest in hormone therapy.

Yours, etc.,

A. TENNANT CHAPPLE.

British Medical Association House,
135, Macquarie Street,
Sydney.
April 10, 1941.

VENEREAL DISEASE.

SIR: I am sure Dr. Bean misunderstands me. Whilst I was unable to give the precise number of men away from the front in March, 1918, I knew that a large number were off duty, to which must be added the number who did not report.

Anyone concerned in army administration knows the number of medical officers, orderlies and supply services that are involved in managing a venereal diseases hospital.

The general German superiority as stated in my work was three to one. The point is that a reserve of men not necessarily in the front line might well have stemmed the rush and given time for reinforcement from England, where a large number of men were available.

The figure given of 400,000 of all troops for the war are not mine, but appear in the Official History of the War, Volume 2, where a very temperate and detailed account of the problem is set out, including the humble part I played. You cannot simply select one day and divide the 400,000 by the number of days in the war and expect to get accurate results.

About outbreaks of venereal disease in the Australian and British armies full particulars are given in two of my works, "A Vision of the Possible" and (with Lieutenant Deane) "The A.A.M.C. in Egypt", together with the active measures taken to control it.

The varied incidences in British and other troops are set out in the Official History.

Whether my *"obiter dicta"*, as Dr. Bean calls it, is sound or not, I am indeed glad that attention has been given to this serious problem in the present war, when every man is wanted. This time the danger is not being underrated.

Yours, etc.,

JAMES W. BARRETT.

105, Collins Street,
Melbourne, C.1,
April 21, 1941.

SOME MEDICAL ASPECTS OF CRIME.

SIR: Dr. Ellery, in his criticism of Dr. Dane's recent letter, refers to the harmful effects of unrecognized emotional needs, especially in those members of the medical profession who have chosen to deal with patients suffering from the same class of disorder.

These effects are serious, in both our clinical and social work.

There is need of a training centre in Melbourne where such conditions in the practitioner could be brought to consciousness and if possible remedied, and where psychotherapists of different schools of conscious thought and different aptitudes might supplement one another's work.

Yours, etc.,

ALICE M. BARBER, M.B., B.S. (Melbourne),
Honorary Assistant Psychiatrist, Children's Hospital, Melbourne; Honorary Psychotherapist, Queen Victoria Memorial Hospital, Melbourne.

71, Collins Street,
Melbourne,
April 22, 1941.

SIR: The recent letters in THE MEDICAL JOURNAL OF AUSTRALIA from Dr. Paul Dane and Dr. Ellery raise questions that are by no means merely of academic interest to the medical profession. The psychiatrist is constantly called upon to deal with criminal tendencies and with the broader problem of moral delinquency, and in many apparently medical conditions the moral aspect is present whether the practitioner recognizes it or not.

To label the criminal as antisocial and thus a menace to society is true enough, but is not the whole truth, for, as Dr. Ellery has pointed out, the criminal is also a product of society, and society, while having the right to protect itself, has also a moral obligation to the criminal.

Society's power to thus defend itself is vested in courts of law, and today the legal profession itself is demanding a more enlightened approach to the problem of the criminal. The judge wants to know not only the results of criminal behaviour, but also the causes of it, an attitude more in line with the modern attitude of the medical profession towards disease.

A person with open tuberculosis is a menace to society, but we do not merely punish such people. We may have to segregate them for the protection of society, but we do it in sanatoria, not in gaols, and the welfare of the patient is also accepted as a moral responsibility by society through its representative, the doctor.

The case of the criminal is quite analogous, and the legal profession is becoming dissatisfied with the results of mere punishment (*vide* statistics in Dr. Ellery's letter). Judges are becoming more therapeutically minded and are looking for a sound psychopathology.

Psychopathology is the study of morbid distortions in the relationships between individuals, and is related to medical pathology and through that to physiology and biology. The medical profession, with its training in these subjects, as well as its tradition of clinical experience, is best fitted to extend its knowledge from the treatment of the isolated individual to that of the individual seen as part of a social structure.

Every neurotic is a potential delinquent, and most sick people have an element of neurosis in their clinical picture. Isn't it time that the medical profession, which has qualifications possessed by no other group, should seriously study the problem of emotional adjustment and give a lead which could be authoritative and would be welcomed by other professions and would render intelligible the problems surrounding neurotic and near-neurotic illness with an enormous increase of interest to ourselves and value to our patients and to society in general?

We must remember that Nature imposes strict discipline; but there is a world of difference between discipline and punishment. The work of the psychiatrist and to a lesser extent of the physician is largely to reveal what are the legitimate disciplines of Nature, and how the attempt to evade these disciplines results in disease, either physical, spiritual or moral. Therapy consists in reconciling the patient to the acceptance of these disciplines, a principle carried out in the practice of hygiene for example. Nature can look after her own disciplines, and society can be relied upon to do all and more than is required in the way of punishment. What the delinquent needs is someone to give him encouragement, understanding, and the inspiration to build up his lost self-respect.

Yours, etc.,

H. SELBY LINK.

61, Collins Street,
Melbourne,
April 25, 1941.

SYPHILIS IN PREGNANT WOMEN.

SIR: Dr. MacKnight states that the only published figures for Australia as regards syphilis in pregnancy are those of Fowler, who found an incidence of 10·7% in women attending the Women's Hospital, Melbourne. She will, therefore, be interested to learn that results resembling her own were published in THE MEDICAL JOURNAL OF AUSTRALIA on December 5, 1936, by T. Dixon Hughes and L. Gammie, who reported the results of routine use of the Wassermann test on 3,404 patients attending for antenatal care at the Women's Hospital, Crown Street, Sydney.

These tests revealed an incidence of 0·47% for *primiparæ* and 1·31% for *multiparæ*. A further report carrying the figures up to the present date is in course of preparation, and from a casual survey the incidence will be slightly below 1%.

Yours, etc.,

T. DIXON HUGHES.

135, Macquarie Street,
Sydney,
April 23, 1941.

Obituary.

FRANK TIDSWELL.

DR. FRANK TIDSWELL, whose death was announced recently in this journal, was one of the pioneers in the practice of pathology in New South Wales. He was known to the older generation of medical graduates as one whose driving force created an effective governmental department in pathology at a time when vision and initiative were lacking. In later years he had been head of the department of pathology in the Royal Alexandra Hospital for Children. He brought to this institution years of experience and a great wealth of knowledge. How effective he made his department is well known to those with whom he associated. The tributes published herewith from Professor J. B. Cleland, Sir Robert Wade and Dr. J. I. C. Cosh show what manner of man he was and will reflect the opinions of many of his former colleagues.

Professor J. Burton Cleland writes:

I am glad to be given this opportunity of expressing my appreciation of the scientific work and administration of my late chief and colleague Frank Tidswell. My associations with him were particularly pleasant and I personally owe

much to the opportunities presented by the Bureau of Microbiology of New South Wales, which was established by the Government at Frank Tidswell's instigation in 1908. All his colleagues appreciated his scientific outlook, his thoroughness and his technical skill. He played an important role in very varied biological directions in the development of New South Wales in the period between the late nineties and the outbreak of the Great War in 1914.

Tidswell first won a reputation by his investigations into plague and its transmission when this disease was introduced into New South Wales at the beginning of 1900. As a house surgeon at the then Prince Alfred Hospital I heard much of the investigations being made at the Central Board of Health. Tidswell had already been associated at the university with C. J. Martin and, I believe, Almroth Wright. These associations enabled him to master the developing science of public health bacteriology and also led him later into investigations into snake venom and the poison associated with the spur of the platypus. He and his chief, Ashburton Thompson, were both rather masterful persons and did not always see eye to eye. Tidswell envisaged emancipation from the control of the Department of Public Health through the establishment of a Bureau of Microbiology, which would include not only activities in connexion with human diseases and public health, but would also deal with diseases of animals and of plants. The idea was an excellent one, and in spite of opposition Tidswell was able to effect its acceptance by the Government. This led to the establishment in July, 1908, of a separate branch of the Chief Secretary's Department, under the title of the Bureau of Microbiology. In the preface to the first report of this body Tidswell gives an account of its origin and of the activities upon which it would be engaged. The bureau was to deal with the parasitic and microbic diseases of plants and of stock and with various farming problems of a microbiological nature. It was also to consider what further steps should be taken to deal with the matter of rabbit destruction, in continuation of the bacteriological work Tidswell had already been engaged in on Broughton Island. These activities were in addition to those of the Public Health Laboratory and of a laboratory serving the then Coast Hospital. Tidswell stressed the point that the organization of the bureau, which had arisen out of the business needs of the community, followed the ideals represented by such great institutions as the Lister Institute in London and the Pasteur Institute in Paris. Research was as important an aspect of its activities as mere bread-and-butter requirements. The Bureau of Microbiology properly started its official career in January, 1909. In 1908 I was holding the position of government bacteriologist and pathologist in Western Australia, and was approached by Dr. Tidswell to accept the position of principal assistant microbiologist in the new bureau, with himself as the director. I took up my duties at the end of January, 1909, and found associated with me as assistant microbiologists G. P. Darnell Smith, eventually director of the Botanic Gardens, A. E. Finckh, T. Harvey Johnston, now professor of zoology in the University of Adelaide, and, later, Burton Bradley. Robert Grant was the senior assistant, carrying with him the traditions of C. H. Martin and Almroth Wright, for whom he had worked. The staff cooperated very harmoniously together, and the first four annual reports up to the end of the year 1913 show what valuable work was done under Tidswell's able administration. In 1912 Dr. Tidswell left on a visit to Europe, and during his absence changes were foreshadowed as the result of the retirement of Dr. Ashburton Thompson. As a consequence Tidswell resigned his appointment as Director of the Bureau of Microbiology in February, 1913, and in the beginning of March that portion of the work of the bureau dealing with diseases of man and animals and public health problems became the microbiological laboratory of the Department of Public Health. The agricultural side was separated off under G. P. Darnell Smith as the biology branch of the Department of Agriculture. There is no doubt that Tidswell's Bureau of Microbiology, with its wide scope of activities, was a wise movement. It brought together workers in allied branches of science who were able to help each other greatly in working out the problems on which they were engaged. Time and again this close daily association proved its value. It was a short-sighted policy to break up this association, which foreshadowed in a way the present Commonwealth Council for Scientific and Industrial Research.

After his retirement from the government service, Tidswell actively continued his activities in bacteriology. The annual reports of the Microbiological Laboratory that followed those of his Bureau of Microbiology emphasize the impetus he gave to microbiological work in Australia. These and his work on plague, on the biological control of the rabbit pest and on snake and other venoms are memorials to him which will be consulted from time to time for many generations.

Sir Robert Wade writes:

In the passing of Dr. Frank Tidswell the Royal Alexandra Hospital for Children has suffered a severe loss; the staff has lost a loved and tried friend and the hospital a most valued servant.

His first association with the hospital was as honorary microbiologist in 1914, in 1919 as honorary pathologist and microbiologist, and in 1925 as director of pathology, and this has been his sole work for the past sixteen years.

With his previous experience at the Board of Health and in private practice he was particularly well qualified to guide the work in a rapidly growing department in what is a large hospital in which the training of students plays a large part.

He was especially interested in the relationship between his department and the clinical side of the hospital work, and laboured hard to establish the necessary liaison; at consultations his wide knowledge and clarity of reasoning would always help to crystallize the picture of the morbid condition in his listeners' minds.

His was an orderly mind, and the pathological museum at the hospital is a monument to this; he arranged the specimens so that they would clearly demonstrate the progress of each condition shown and the variations; nothing confusing or redundant was allowed on the shelves; so for the student there was defined a clear picture of each pathological condition.

Perhaps his greatest value to the medical staff of the hospital was shown at the monthly clinical meetings; his notes on the pathological findings were always clear and illuminating, and these meetings would generally end with a lecturette from him on some subject in which he showed himself to be fully acquainted with all the recent work and thought. He was much more abreast of all recent literature than, I am afraid, were we, his colleagues. He was a clear thinker, young in mind and always receptive of new ideas.

As an administrator he excelled, and his department ran like clockwork: in him his subordinates found a good friend, generous to all, unstinting in his help when asked.

His gentleness of manner, his quiet courtesy and humour, endeared him to all his colleagues, and the hospital has lost a most valued specialist in his own department.

Dr. J. I. C. Cosh writes:

Since my retirement from practice to live at Moss Vale an acquaintance with the late Dr. Frank Tidswell, going back some forty years, ripened into a close friendship. It is to the memories of my friend during this later period that I would like to pay tribute.

Meeting him at his country home, Farnborough, one was struck by the changes time had made since my early recollection of him, still full of energy, however, as he worked in his garden. Strolling round amongst the beautiful old trees, his talk was full of interest, derived from his great fund of general knowledge—trees, flowers, soils, draught-horses, snakes *et cetera*, and so to the kennels; somewhere here one usually came on Mrs. Tidswell busy with the Scotties.

The doctor was a keen motorist. In a shed near by stood his 1912 Rolls Royce. I think that nothing would have induced him to part with it but the increasing tax on his strength when changing a huge tyre on the road, and the restrictions on petrol. A rest in his well-stocked library disclosed his taste in books, the while he talked on all sorts of subjects, and so ended a happy morning.

Recently I had the pleasure of being shown over the pathology department at the Children's Hospital. With justifiable pride he drew attention to the varied equipment and explained the working of his system of case records—"all that is essential and nothing not needed" he summed it up. Parting with him that day, there was nothing to suggest that so soon would the place thereof know him no more.

He enjoyed his golf and was looking forward to another game when I saw him last.

Were I asked what it was about the doctor that appealed to one, I should without hesitation say his gentle manner, his kindliness and consideration, with a strong determination and intolerance of any "not playing the game". To those who knew him in his work or in his leisure there will always remain the memory of a very courteous gentleman.

LEONARD GUSCOTTE HAYDON.

WE regret to announce the death of Dr. Leonard Guscotte Haydon, which occurred on April 27, 1941, at Melbourne, Victoria.

REGINALD HERBERT MORRISON.

WE regret to announce the death of Dr. Reginald Herbert Morrison, which occurred on May 1, 1941, at Melbourne, Victoria.

THE MEDICAL WAR RELIEF FUND.

THE following is a second list of contributions to the Medical War Relief Fund established by the Federal Council of the British Medical Association in Australia for the relief of distressed medical practitioners in Great Britain.

Western Australia.

£10: Dr. J. E. McMahon.
£5 5s.: Dr. G. W. Barker, Dr. E. R. Dermer, Dr. W. H. Nelson, Dr. L. D. Hodby, Dr. I. Thorburn, Dr. J. P. Ainslie.
£5: Dr. Thelma Robinson.
£2 2s.: Dr. D. Gawler, Dr. F. G. Stimson, Dr. N. M. Cuthbert, Dr. F. H. U. Baker, Dr. M. F. Williams.
£1 5s.: Dr. A. B. Wilson, Dr. J. V. Arkle.
£1 1s.: Dr. G. A. Kelsall, Dr. A. R. Bean, Dr. A. E. Williams, Dr. S. L. Harke, Dr. O. N. Walker.
10s. 6d.: Dr. W. Muir, Dr. J. J. Savage.

The Royal Australasian College of Surgeons.

MEETING OF BOARD OF CENSORS.

THE next meeting of the Australian Board of Censors of the Royal Australasian College of Surgeons will be held at the College, Spring Street, Melbourne, probably in September, 1941. Candidates who desire to present themselves at this meeting should apply to the Censor-in-Chief for permission to do so on or before June 30, 1941. The appropriate forms are available at the College, Spring Street, Melbourne, and at the offices of the various State secretaries.

Notice.

THE University of Melbourne announces that a course of instruction in Part I of the subject for the Diploma of Diagnostic Radiology has been arranged and will begin in the week commencing May 12, 1941. Candidates who desire to take the course should lodge enrolment cards with the registrar immediately.

THE annual general meeting of the Australian Army Medical Corps Officers' Mess, Eastern Command, will be held on Saturday, May 24, 1941, at the Imperial Service Club, Sydney, at 7.30 o'clock p.m. Combined with this meeting there will be a complimentary dinner to Major-General F. A. Maguire, C.M.G., D.S.O., V.D., on the occasion of his appointment as Director-General of Medical Services. The subscription will be 10s. 6d. Uniform, field service or black tie. Members of the Returned Medical Officers' Association are asked to accept this as a notice to attend on that date. Those who intend to be present are asked to notify the Honorary Secretary at the office of the Deputy Director of Medical Services, Sydney, by Wednesday, May 21.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Dunlop, Leslie Critchley, M.B., B.S., 1940 (Univ. Sydney), Sydney Hospital, Sydney.
Allsopp, Raymond Jesse, M.B., B.S., 1939 (Univ. Sydney), 61, Kembla Street, Wollongong.
Collett, Frederick Joseph, M.B., B.S., 1939 (Univ. Sydney), 1, Fuller's Road, Chatswood.
Hodgkinson, Henry Richard Taylor, M.B., B.S., 1939 (Univ. Sydney), 44, Anson Street, Orange.
Thomas, James Frederick Noel, M.B., B.S., 1937 (Univ. Sydney), 153, Wycombe Road, Neutral Bay.

Diary for the Month.

MAY 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 13.—Tasmanian Branch, B.M.A.: Branch.
MAY 15.—Queensland Branch, B.M.A.: Ipswich Hospital Clinical Society.
MAY 15.—Queensland Branch, B.M.A.: Hospital for Sick Children Clinical Society.
MAY 15.—Victorian Branch, B.M.A.: Ethics Subcommittee.
MAY 19.—Victorian Branch, B.M.A.: Hospital Subcommittee.
MAY 20.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 20.—Victorian Branch, B.M.A.: Organization Subcommittee.
MAY 20.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
MAY 21.—Western Australian Branch, B.M.A.: Branch.
MAY 22.—New South Wales Branch, B.M.A.: Clinical.
MAY 22.—Victorian Branch, B.M.A.: Executive.
MAY 23.—Queensland Branch, B.M.A.: Council.
MAY 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 28.—Victorian Branch, B.M.A.: Council.
MAY 29.—New South Wales Branch, B.M.A.: Branch.
MAY 29.—South Australian Branch, B.M.A.: Branch.
MAY 30.—Queensland Branch, B.M.A.: Commencement of Post-Graduate Course.
MAY 30.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Proserpine District Hospital; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

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